

# Journal OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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## *Correspondence*

June 13, 1957

Dear Dr. Aitken:

That portion of the editorial in the April 1, 1957 *J.A.V.M.A.* (p. 315), "Symptoms vs. Signs," interests me. I would appreciate an expression from you on this. The question arose in 1952 when Brandy used "signs and symptoms" in the chapter on Newcastle disease in "Diseases of Poultry." I accepted it—my leaning is toward "flexibility of expression" within reason. Is this based merely on a definition by a dictionary author which is taken up as a fact? In this case does not long usage warrant the acceptance of the broad term "symptoms"? This still leaves the option to indicate whether they are objective or subjective. I do not like the word "sign." It suggests to me the signs for degrees, inches, feet—and a piece of board on which may be painted entrance, exit, etc. If sign is accepted—is it possible to prescribe symptomatic treatment? Then must we accept "signology" to indicate a study of signs?

It seems to me that Dorland's definition of symptom is acceptable—"any functional evidence of disease or of a patient's condition"—and is a workable one. It suggests to me any evidence that can be obtained while the animal is still living, i.e., the clinical picture.

I don't like to be stampeded by those who are ready to accept anything new without careful evaluation, e.g.: streptococcosis vs. streptococciosis—the latter is not correct on etymological grounds; the suffix "osis" should be added to the root and not to the adjectival form. Parrakeet vs. parakeet—Mayo and California medical editorial policies differed. About that time Webster's late edition appeared in which they used the single "r." I inquired of the Merriam Company on what etymological ground they based the change—is it "para" to indicate "like," i.e., a "keetlike" bird and is the double "r" based on the French *perrot*. The illuminating reply stated that of seven or eight sources consulted, five used the single "r"—suggestive of the Gallup poll method.

I am somewhat inclined to stay with symptoms, symptomatology, etc. I realize you merely stated the case, but some individuals grabbed it like a Supreme Court verdict. What is your opinion and what policy will you adopt?

Sincerely,  
S/H. E. Biester,  
Ames, Iowa

[We prefer the term "sign" to indicate objective evidence of disease (those "discoverable" by an observer), fully realizing that its use introduces some complications. However, since this is not yet a firm change we will defer to the preferences of authors.—ED.]

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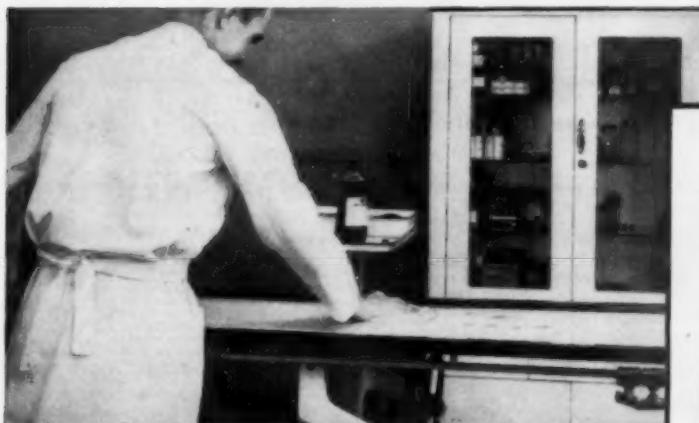
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# AVMA ★ Report

## Dr. Russell E. Rebrassier Chosen President-Elect at Cleveland



Dr. Russell E. Rebrassier

A long-time faculty member of the College of Veterinary Medicine at the Ohio State University, Dr. Russell E. Rebrassier (OSU '14) of Columbus, Ohio, was elected to the office of president-elect at the Ninety-Fourth Annual Meeting of the AVMA in Cleveland, August 19-22.

After spending a year with a commercial antitoxin laboratory, he became instructor and professor of veterinary parasitology, College of Veterinary Medicine, at Ohio State University, in 1916, and was appointed chairman of the department in 1939. He served in this capacity until 1955, when he was made assistant dean of the College of Veterinary Medicine. He has written many articles on veterinary parasitology.

He became a member of the AVMA Council on Education in 1951 and has served as secretary of the Council since that time.

Secretary of the Ohio V.M.A. from 1929 until 1946, he was elected president in 1949. He and Mrs. Rebrassier reside in Columbus.

### Radio and Television Programs Featuring AVMA Members and the 94th Annual Meeting in Cleveland

This is a partial list of programs broadcast before and during the Cleveland Convention, or tape-recorded for rebroadcast at later dates.

**WGN-TV (Chicago)**—Half hour on "RFD Chicagoland" broadcast, August 10, at 11:30 a.m.

Subject: The AVMA and Its Annual Meeting.

Speaker: Dr. John G. Hardenbergh.

Subject: What the Veterinarian Does for the Farmer.

Speaker: Dr. Richard E. Tully.

Demonstration: Care of the Bovine Foot.

**WBBM-TV (Chicago)**—Eight-minute interview on "Town and Country" broadcast, August 12, at 6:45 a.m.

Subject: The Purpose of the AVMA Convention.

Speakers: Drs. Harry E. Kingman, Jr., and A. Grant Misener.

**WBBM (Chicago)**—Five-minute tape recorded interview for broadcast on "Farm Hour," August 14, 6:45 a.m.

Subject: The AVMA and organized veterinary medicine.

Speaker: Dr. Harry E. Kingman, Jr.

**WBBM (Chicago)**—Five-minute tape recorded interview for broadcast on "Farm Hour," August 16, 6:45 a.m.

Subject: AVMA Organization and Veterinary Medicine in Illinois.

Speaker: Dr. A. Grant Misener.

**WDCK (Cleveland)**—30-minute tape recorded panel for broadcast on the "Bill Reid Show," 6:15 to 6:30 p.m., August 19 and August 20.

Subject: What Do Veterinarians Do?

Speakers: Drs. Wendt, Rebrassier, Rickards.

(Continued on adv. page 53)

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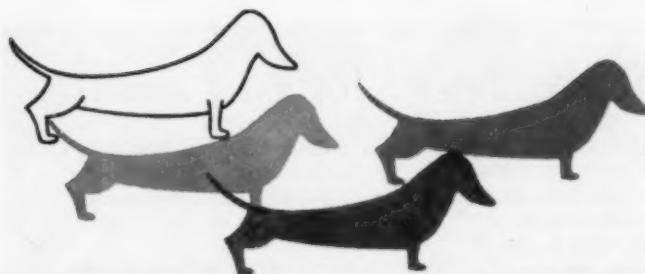
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1. Rachman, M., and Frucht, T. R.: Vet. Med. 49:341, 1954.



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REFERENCES: 1. Bull, W. S., N. Amer. Vet., in press. 2. Henry, R. T., and Blackburn, E. G. J. Vet. Med., in press.

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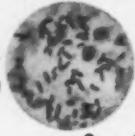
\*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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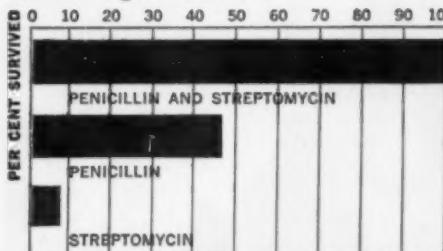
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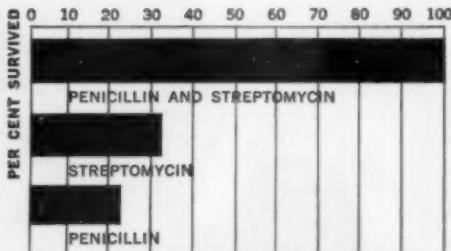
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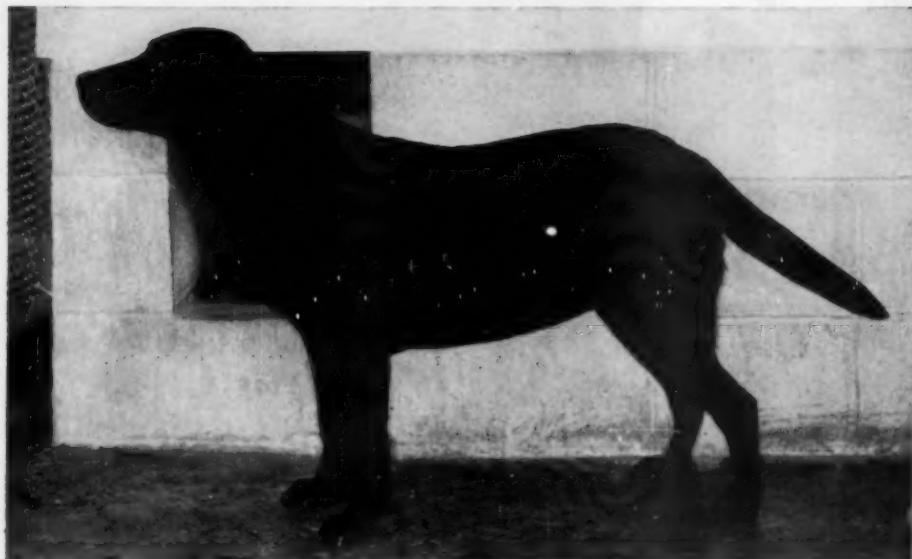
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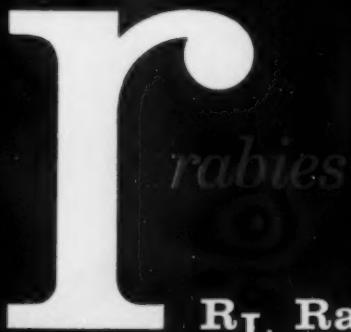


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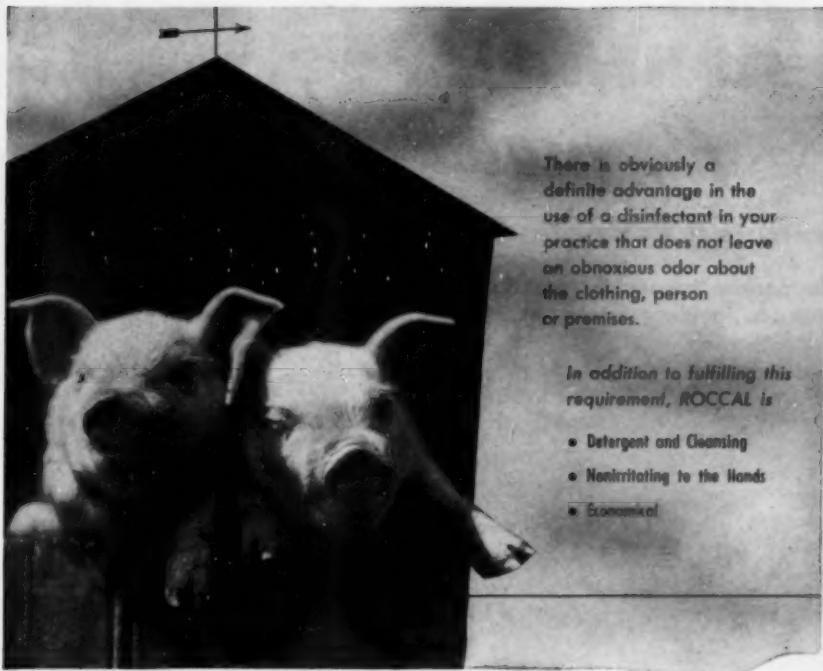


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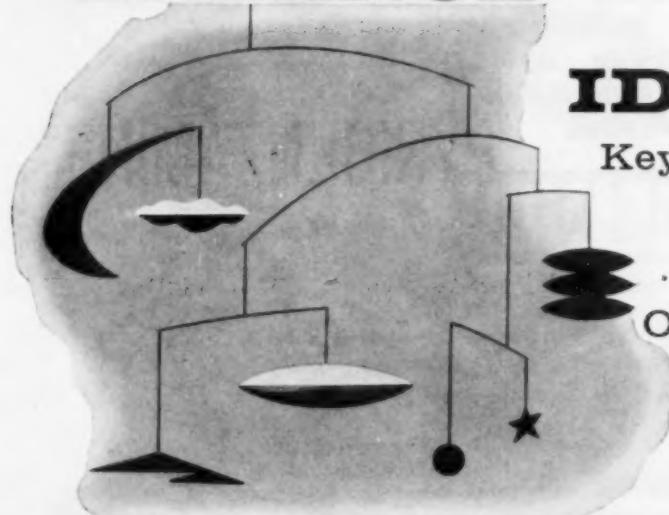
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\*Chambers, E. E.: N. Am. Vet. 37:105 (Feb.) 1956.

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1. Harris, J.R., and Clarkson, T.B., Prevention of Relapses in Milk Fever, *Vet. Medicine*, 12:696 (Dec. 1955).

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## Origin of Hog Cholera

R. P. HANSON, Ph.D.

Madison, Wisconsin

ACCOUNTS of the origin of hog cholera in the United States warrant review as we give serious consideration to its eradication. No other disease of swine has produced such a high mortality, or has been so dreaded, and yet less than 150 years ago hog cholera was unknown.

### FIRST REPORTS ON HOG CHOLERA

Veterinary writers have placed the first appearance of hog cholera in Ohio in the year 1833. Their authority is a document entitled "The Introduction and Spread of Hog Cholera in the United States," published in 1888 as part of the Fourth and Fifth Annual Report of the Bureau of Animal Industry.

This paper analyzes that little-read document of 110,000 words which was compiled from letters of 1,000 correspondents of the U. S. Department of Agriculture. It is not surprising, considering its heterogeneity, that no one has attempted a real summary of the conflicting information. Many may question whether anything can be accomplished by re-examining, after 80 years, statements made by a diverse group of men, few of whom had any scientific training. However, others point to the productive ideas experimentalists have obtained from the shrewd observations of livestock men. Whatever the viewpoint, re-evaluation should help to separate fact and folklore, and perhaps it may stimulate research in epizootiology of hog cholera.

The report's most important point, that hog cholera is of recent origin, has been

accepted rather generally. However, the year (1833) and the place of origin (Ohio) were determined by editorial selection<sup>1</sup> and not by evidence, as the claim can not be substantiated from the published extracts of letters to the Bureau. The only tests of validity that we can now impose on the reports of correspondents are their inherent consistency and their agreement with other accounts from the same region. When the writer described hog cholera as a pustular disease or one with a 10 per cent mortality, he probably was referring to another disease. When he referred to prior outbreaks of hog cholera in another county, or when he claimed that the disease was present in his county 25 years before any of the counties within 50 miles of him had recognized it, the report is inconsistent with a claim of priority; also inconsistent with the behavior of the disease which would not be expected to remain confined to an area of a county for years.

The compiler of the Bureau of Animal Industry document enumerated ten reports of a disease presumed to be hog cholera which occurred prior to 1846, listing the state, but not identifying the county in which the disease occurred. Seven of the ten are readily identified, the eighth was incorrectly listed, and two, one from Illinois in 1840 and one from New York in 1844, could not be found. However, four other claims were located.

Quotations from nine of the first reports will give the reader a basis for judgment. The earliest report among those in the compilation was from Franklin, Tenn., and would place the outbreak about 1810. Three counties within 50 miles of Franklin County reported hog cholera before 1850

From the Departments of Veterinary Science and Bacteriology, University of Wisconsin, Madison.

Published with the permission of the director of the Wisconsin Agricultural Experiment Station as paper NS 229.

and 16 reported it by 1860. The correspondent<sup>2</sup> wrote, "The disease known as hog cholera has prevailed more or less in this county since the first settlement, although not called by that name here until within the last twenty years." The county was settled between 1800 and 1810.

A hog cholera-like disease was said to exist in Tangipahoa, La., in 1818, 70 years before 1888. No other counties within 50 miles of Tangipahoa had reported the disease by 1850 and only two by 1860. The correspondent<sup>3</sup> wrote, "The oldest citizens remember hogs dying as they do now 70 years ago. Nothing is known as to the introduction of the disease."

The third report, the one credited by the compiler as describing the first appearance of hog cholera in America, came from Muskingum, Ohio. However, no other county within 50 miles of Muskingum had reported the disease before 1850 and only one other by 1860. The correspondent<sup>4</sup> himself did not claim that the 1833 outbreak in Ohio was the earliest. "Hog cholera

was first known in this county in 1833. Some few died on the Muskingum River from May to December. Its appearance was again made in 1842, also in 1849-1850 to 1855. At no time was it so bad as on the Wabash River, Indiana, in 1830 to 1833, 1840 to 1845, up to 1870 it increased nearly 10 per cent per annum."

A correspondent from Lexington, S. Car., reported the disease in 1837. Hog cholera was not reported within 50 miles of Lexington in the next 13 years but, by 1860, seven counties within 100 miles had experienced the disease. In the words of the reporter,<sup>5</sup>

When a small boy over 50 years ago there were similar epidemics among hogs to that now known as cholera. It is possible that the closer confinement which had followed the stock law, and the stuffing process in feeding may have aggravated the diseases which have always attended the raising of hogs; for I am satisfied that this scavenger is more or less diseased on account of the hogish habits and the foul food upon which it subsists. There has been a terrible visitation of the cholera, so-called among the fattening hogs of this locality during the past month, and many have died.

Hog cholera was reported from Fayette, Ky., in 1840. Five other counties within 50 miles recognized the disease before 1850 and 26 counties within 100 miles reported the disease by 1860. This is a convincing substantiation. The correspondent<sup>6</sup> wrote,

The year of the first appearance of hog cholera in this county and mode of its introduction cannot be definitely stated. Somewhere in the years from 1840 to 1848 the hogs of the writer were severely attacked by it, first commencing in young pigs, then older hogs, sows in pig would bring forth their litter all dead, the sows also dying, the losses all told amounting probably to more than 100. Each fall for several years I had visitation of it; had heard of the disease for several years before it visited me; have had nothing of the kind for many years, attributable to the fact, as I think, that now I keep but few hogs which have a large range on the farm.

Hog cholera was reported from Harrison County, Indiana, the same year, 1840. By 1850, one other county within 50 miles of Harrison had reported hog cholera and, by 1860, 32 counties within 100 miles had been visited by the disease. The correspondent<sup>7</sup> stated, "Hog cholera made its appearance in this county about 1840. We did not know any cause for it. Hogs previous to that time were healthy."

Although cited by the compiler, the dis-

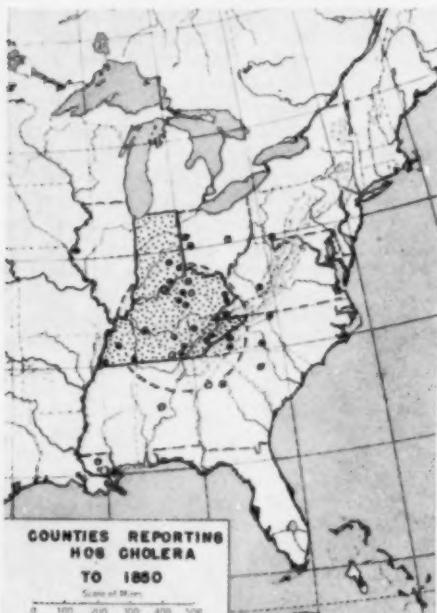


Fig. 1—Reports of hog cholera to 1850. Counties reporting hog cholera 1810 to 1840 (o). Counties reporting hog cholera 1841 to 1850 (•). Stippled states had highest hog population in 1850. Circle (formed by dashes), with a radius of 200 miles, encompasses about 70 per cent of the report.

ease described by the correspondent from Clinton, Ala., in 1840, would not appear to be hog cholera. The disease had appeared only twice within 100 miles of the county by 1860. In the words of the contributor,<sup>8</sup> "Hog cholera has existed in this country since 1840 to my knowledge. Hogs that are well fed and given proper attention scarcely ever have it; but those that run at large and get poor during winter and spring and then are put on oat pastures generally contract the disease. It is only widespread and fatal when no attention is given animals."

The fourth report<sup>9</sup> of the disease in 1840 was from a "Greene County." Although entered in the Florida section, there was not and is not a county of that name in Florida. Four of the eight states that have a Greene county submitted reports credited to that county. This left four states—Indiana, Missouri, North Carolina and Ohio—as possible contenders for the "orphan" county. The Greene counties in Missouri and North Carolina are far from other early outbreaks, but the Indiana county on the Wabash River, and the Ohio county on the Little Miami River are both in areas from which there were many other early accounts of hog cholera. "We call every disease that attacks hogs and they die in large numbers hog cholera. The disease that prevailed in my county the past

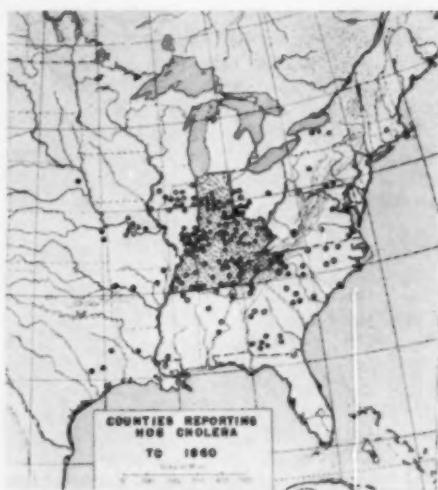


Fig. 2—Reports of hog cholera to 1860. Counties reporting hog cholera 1810 to 1850 (o); counties reporting hog cholera 1851 to 1860 (•).

year was not precisely the same as the cholera of 12 or 15 years ago. Not so many die now as then according to the number attacked. The disease last fall was more confined to pigs than to hogs of 18 months and 12 months, although a good many of the latter died. The date when cholera first



Fig. 3—Reports of hog cholera to 1890. Stippled states had highest hog population in 1890.



Fig. 4—Reported sources of initial infection. Arrows indicate the states from which the infection was alleged to have come and the ones in which the disease occurred. Arrows entirely within a state indicate direction of movement of the disease within the state.

made its appearance is not known to your correspondent, but many years before 1860—probably forty or fifty years ago." Since this was written in 1887, that would be 1847 or 1837.

All of the reporters are rather vague about the date, since it is based on their memory of an event 40 to 50 years earlier, apparently unsupported by records or diaries. However, there was no serious contradiction in three of the four accounts for 1840, and the counties adjacent to that of the reporter confirmed the presence of the disease in the decade that followed. The region in which hog cholera first was recognized can be encompassed by a circle with a radius of 200 miles, having its center in Clay County, Tennessee (fig. 1). It includes most of Kentucky and Tennessee and areas of Indiana, Alabama, Georgia, and North Carolina. Whether or not the point is of major significance, this was the region in which the swine population was heaviest in 1850. The later geographical distribution of the disease and of the swine populations are shown (fig. 2, 3; table 1).

Apart from determining the location of the earliest appearance of hog cholera, another purpose of the 1888 report was to determine its origin. The compiler<sup>1</sup> of the report seemed satisfied "that the contagion was imported from Europe with some of the animals that were brought from there to improve our breeds of swine." European

TABLE I—The Population of Swine and Reports of Hog Cholera in 1850 and 1890\*

State	No. pigs per sq. mi.		No. counties	BAI Report of 1888		
	1850	1890		No. counties replying	1850	1887
Tenn.	74	55	95	55	7	55
Ky.	71	55	120	54	6	54
Ind.	62	79	92	50	2	48
Ohio	40	54	88	43	3	28
Ga.	37	28	159	64	2	64
Ala.	37	30	67	24	1	24
Md.	35	34	23	10	0	10
Ill.	34	97	102	55	2	55
S. Car.	34	21	46	11	1	11
N. J.	34	29	21	9	0	9
Miss.	33	30	82	25	0	23
N. Car.	31	24	100	51	3	51
Va.	27	25	100	42	1	42
Mo.	24	73	114	54	0	54
Pa.	23	26	67	22	1	10
N. Y.	20	13	62	23	0	5
R. I.	19	13	5	2	0	2
Conn.	15	4	8	5	0	3
Ark.	15	31	75	28	0	26
La.	12	14	62	20	1	18
Mass.	10	8	14	4	0	4
Iowa	5	103	99	53	0	51
Wis.	3	19	91	35	0	20
Mich.	3	16	83	35	0	9
Texas	3	9	254	61	0	30
Fla.	3	6	67	17	0	14
Maine	1	2	16	9	0	2
W. Va.	9	20	55	26	0	22
Kan.	0	31	105	50	0	34
Neb.	0	29	93	43	0	34

\*Sixteen other states and territories with small swine populations were omitted from the table; †Part of Virginia.

authorities have disagreed and have maintained that evidence of prior occurrence of hog cholera in Europe was not uncovered.

Before pursuing the question of origin, we should distinguish carefully between the disease produced in a certain host at a given time and the parasite which induced it. For there to be a disease, there must be a host as well as a parasite and they must meet under certain conditions. A disease, in other words, is an event rather than a living entity and the participants in the event may exist without the occurrence of the event, as baseball players can live without meeting and can meet under some conditions without playing ball. So a host and a parasite may exist in the same region without meeting, or may meet without a serious disease resulting. The latter is not unusual with many pathogens. The resistance of a host to its parasite may be high because of genetic traits, or because of special nutrition, or altered environmental conditions. The extent of the change in the host from one generation to another or one season to another may be such that manifestation of disease is seen in one instance and not in the other. One need not turn to spontaneous generation of parasites to explain the origin of a new disease. One need only study the equilibrium between the host and parasite.

#### ETIOLOGY

There are at least three explanations for the origin of hog cholera during the early nineteenth century. Each had adherents among the contributors to the 1888 report; and since that time, we have uncovered a little evidence for all three theories but not much evidence for any one of them.

1) *The New Versus the Old Hog Theory*.—The virus of hog cholera may have existed in swine long before the disease was recognized in its present form because before that time: (a) the swine were genetically more resistant, (b) the swine were more refractory because of their diet, (c) the swine were less susceptible because of environment.

2) *Reservoir Theory*.—The virus of hog cholera existed in another species of animal (a reservoir) from which swine, through association, became infected and once infected could infect other swine.

3) *Importation Theory*.—The virus of hog cholera existed in swine in another region where, because of the poorly developed husbandry, the disease was not recognized and from which the disease was imported.

That the hog of the pioneer was immune to hog cholera by virtue of innate or

genetic refractoriness was a favorite theory among swine breeders. The razorback, the common hog of the frontier was not supplanted by improved breeds from Europe until sometime after the middle of the nineteenth century. During this period, there was ample opportunity for observation of real or fancied differences in susceptibility. A typical contributor<sup>10</sup> claimed, "Our full native stock is not affected at all or at least I have not been able to learn of an instance. Our hogs were healthy before we began to breed up, but were of a very poor grade." Others<sup>11</sup> qualified their statement. "The disease does not affect the old stock of hogs, the elm peelers, as badly as the more improved kinds." This, they<sup>12</sup> felt, could be ascribed to the constitutional and physical difference in the old and improved hogs. "It required two years to get the woods stock ready for market. The improved hog is fat from a pig and ready for market at any age." Some<sup>13</sup> went so far as to say, "My opinion is that we must retrace our steps in the hog breeding business or be constantly subject to disease."

So many changes were occurring in swine husbandry that it was not easy to separate the effects of breeding, diet, and management. This is clearly stated by a correspondent<sup>14</sup> from Indiana, "There has been such a change in the last 25 years in raising hogs that it has affected the animals' constitution. It used to be that a hog was wintered once before he was fattened. Now he is crowded on a corn diet from a pig, which makes him susceptible to cholera. The greater the variety a hog has in his food the more healthy he will be, and the older a hog is the better he can stand a corn diet." The inadequacy of corn diet and the need for variety was stressed by many contributors. A few<sup>15</sup> linked the appearance of hog cholera to introductions of plants, such as sorghum or red clover, or the appearance of rust on oats. "Its cause was generally attributed to the introduction of sorghum cane. It was discovered on those large plantations where sorghum was first grown." Contributors from many states reported a relationship between abundance of "mast" (acorns and nuts) and hog cholera. A writer<sup>16</sup> from Arkansas put it in these words, "Nearly every year we have more or less mast and after a good mast the cholera is worse. I attribute a good deal to the carelessness of the owner. When a mast gives out, it is

then the hogs need some preventive, but they do not receive it."

Management was believed to play a role. Most important were the changes that came with intensive husbandry; particularly the concentration of many hogs in a small area. "The country being new and sparsely inhabited, hogs measurably subsisted on the range. They were healthy, industrious, and self-sustaining. But when the improved breeds were brought into the country, supported on grain, and confined to enclosures cholera began to make its ravages among them."<sup>17</sup> "There is little or no loss by the disease to small lots raised and fattened on the same farm. It is where large lots are indifferently picked up by feeders and fed exclusively on corn where the most damage is done and that happens only when corn is plenty."<sup>18</sup> There was considerable agreement on the greater hazard with large herds. "Hogs in large droves seem to be the ones that suffer most." Since many good producers were large breeders, observers<sup>20</sup> concluded, "The rule has been that the entire herd goes when attacked and often it is the herd of the most successful feeder thereabouts."

Most epizootics of hog cholera could be traced to direct contact with infected pigs. This was well stated by a correspondent<sup>14</sup> from Indiana. "It starts generally from some weighing place, or from where the drovers stop over night with their hogs and spreads by contact over the neighborhood. I knew a boar to break out and was gone some time and finally came back with the cholera. I knew a sow to wander three miles from home and spread the disease. The last time I had it in my hogs, it was caused by bringing a sow to my boar. It is often spread by not burying those that have died."

Some observers<sup>21,22</sup> believed that all epizootics could not be explained by direct contact. "The buzzards are exceedingly numerous and no doubt carry it from point to point with ease." "There is plenty of proof that crows will spread the disease by means of the parasite adhering to their feet, legs and feathers. During the summer months when they are nesting the disease spreads but slowly. In the fall they flock in countless numbers to pens where hogs are fed, and soon it spreads with increased rapidity. Last fall there were but few crows and but few cases of plague in the counties. This fall they are very numerous

and many are losing large numbers of hogs." Insects were also blamed.<sup>23</sup>

In 1865 there was a visitation of a fatal disease among fed swine which was called cholera. There was a fearful visitation of the buffalo gnats that spring, in April. In the early summer of 1875 there was a mild visitation of cholera and also preceding this the buffalo gnat which drove our team from the fields. In 1844 the most fatal and universal epidemic of hog cholera occurred, which almost destroyed the stock of hogs with us. This followed quickly a severe and prolonged visitation of the gnats. Before our stock had been restored and built up from this, again the cholera (very fatal) visited us and reduced our stock to a minimum so that no farmer made his own meat last fall (1886). The inevitable gnat came on us in March 1886, in dense swarms lasting four weeks. Three days after I noticed the gnats my hogs began dying. These may be coincidences, but I am firmly persuaded that cholera depends upon the poison infused into the system by the bite of the buffalo gnat—I mean the fatal epidemic disease that has carried off our hogs for the past twelve or fifteen years. Of course, I am aware that hogs have died in enormous quantities where the gnat never appeared.

This appears to be a belief not only in a vector but also in an arthropod reservoir of hog cholera. A clear concept of a disease reservoir was still unknown at that time but the precursor idea seems to have been discussed. A correspondent<sup>24</sup> wrote desirively, "Some cranks claimed that the disease was introduced by Norway rats." The reports of spring and fall appearances and the initiation of the disease in lowland habitats are suggestive of the requirements of a reservoir host rather than of the virus. "The disease seems more apt to attack hogs which run upon the commons and along the creek bottom and swamps."<sup>25</sup> Frequently, reporters declared that pigs on the upland and mountain areas were free.

The anthropomorphism of man is evident in the names he has given to diseases of swine. In this animal, he has seen the reflection of his own aches and pains and he has named the diseases accordingly; quinsy, sore throat, measles, cholera, and flu. Obviously, an appellation would be well known for a disease of man before it was given to a disease of animals. Asiatic cholera reached the Ohio Valley in 1833 after invading North America for the first time a year earlier. Thousands of people died in the towns and villages along the Ohio River. A vivid memory of the event re-

mained with the survivors and the reappearance of the disease in 1849, 1866, and 1873 in all parts of the Middle West hog country did not permit anyone to forget.

With this background, it is not surprising that any epizootic disease causing sudden death in swine would be called hog cholera. This was pointed out by correspondents,<sup>26,27</sup> "How it got among our hogs, I never knew; it appeared to strike them like Asiatic cholera strikes man."

"Our people had the cholera in 1833, and I do not think it was many years afterward before the hogs had it; but we have no statistics by which we can arrive at anything like accuracy."

The significant point is brought out by a correspondent<sup>28</sup> from Kentucky. "Forty years ago (1846) hog cholera was not heard of, but the hogs died frequently of a disease called quinsy; the old hogs being scarcely ever affected with it. We once lost 100 pigs from a lot of 105 with quinsy." The implication is that, before the term "hog cholera" was coined, hogs died of a similar disease for which a prior name was used. The crucial point then is whether a similar disease existed before 1840 and not whether a disease called hog cholera existed. Most writers claim few, if any, diseases existed in the early days, but this was not entirely true as other men<sup>29,30</sup> recalled several maladies.

Previous to that time (1854) we had the old breed generally known as the elm peeler or third row hog. There was no disease among them except what was known as measles and kidney worm. In the first disease, the skin bore red specks. It did not often prove fatal. In the second case the animal would circle around, could not go forward in a straight direction, would take a bite of feed and then turn around. This disease proved fatal in about every case.

The controversy over the susceptibility of razorback hogs as compared with the improved hog, or the hog of 1840 as compared with the hog of 1860, might lie primarily in the matter of husbandry. Losses among uncounted pigs in the woods are not as easily recognized as they are among counted pigs in pens. Reasons why the correspondents claimed that disease did not exist in hogs prior to the appearance of hog cholera could be: (1) Some claimed in 1886 that disease of any form did not exist and never had existed in hogs in their counties; (2) it is possible to overlook sporadic deaths, particularly when the ani-

mals are ranging in wild pastures throughout the year; and (3) one tends to forget the difficulties of the old days. Men became more acutely aware of disease when animals were seen daily and the losses were counted.

All of these things—the genetic change in hogs, the change in feeding and managing of hogs, the increased concentration of hogs, the increased movement of hogs to market and for breeding, the increased interest in husbandry, the greater care in counting, and the introduction of a new disease term "cholera"—support the first two suppositions that hog cholera did not appear suddenly in the middle of the nineteenth century; rather that the disease had existed in hogs or some reservoir and had appeared sporadically in the acute form without special recognition until about 1840.

Can the third possibility—importation from abroad—be dismissed? Only one correspondent<sup>31</sup> directly alluded to it. "Some think it was brought into the country by the purchase of hogs from vessels for the purpose of getting new breeds." The early prevalence of the disease in the Ohio Valley, rather than along the Atlantic seaboard, is an argument against the importation of acute hog cholera (fig. 4). However, unless the original reservoir was and is in the United States, hog cholera must have been introduced from overseas by importation of chronic carriers. This presupposes that hogs, at the time of importation, were fairly resistant and losses were not great. There was evidence of healthy carriers among survivors of acute outbreaks. In some instances,<sup>32</sup> groups of carriers were reported. "One farmer bought 40 hogs, 20 from one man and 20 from another. Neither had the disease among his herds. The two lots were put together. All of one lot died, while none of the other were affected by the disease."

#### CONCLUSION

About all that one can conclude from the "Introduction and Spread of Hog Cholera" is that the history of hog cholera should be re-examined with attention to records of that period and to modern thought. The acute disease was recognized in Kentucky, Tennessee, and Indiana during the early 1840's, but the virus may have existed in swine of that area as an atypical disease for many years. The origin of the acute

disease could be more a matter of change in the genetics, nutrition, and husbandry of hogs than an alteration of the virus. Certainly at no time in its history was the hog subject to more changes than during the nineteenth century. Whether the virus was transferred to the swine from an American reservoir animal at some earlier day or was imported with swine from abroad is unanswered by the document.

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All references are to sections of a report by the Bureau of Animal Industry: Introduction and Spread of Hog Cholera in the United States. Fourth and Fifth Annual Reports of the Bureau of Animal Industry for the years 1887 and 1888. Government Printing Office (1889): 187-305.

- <sup>1</sup>Introduction, 187-189.
- <sup>2</sup>Tennessee, Franklin, 281.
- <sup>3</sup>Louisiana, Tangipahoa, 242.
- <sup>4</sup>Ohio, Muskingum, 274.
- <sup>5</sup>South Carolina, Lexington, 279.
- <sup>6</sup>Kentucky, Fayette, 235.
- <sup>7</sup>Indiana, Harrison, 218.
- <sup>8</sup>Alabama, Chilton, 191.
- <sup>9</sup>Florida, Greene, 202.
- <sup>10</sup>Missouri, Laclede, 254.
- <sup>11</sup>Indiana, Cass, 216.
- <sup>12</sup>Indiana, Decatur, 217.
- <sup>13</sup>Missouri, Sullivan, 257.
- <sup>14</sup>Indiana, Huntington, 219.
- <sup>15</sup>Georgia, Early, 205.
- <sup>16</sup>Arkansas, Poinsett, 196.
- <sup>17</sup>Alabama, Franklin, 192.
- <sup>18</sup>Illinois, Clinton, 210.
- <sup>19</sup>Illinois, Stark, 215.
- <sup>20</sup>Illinois, Marshall, 213.
- <sup>21</sup>North Carolina, Hyde, 269.
- <sup>22</sup>Nebraska, Washington, 262.
- <sup>23</sup>Tennessee, Tipton, 286.
- <sup>24</sup>Ohio, Miami, 274.
- <sup>25</sup>Illinois, Johnson, 212.
- <sup>26</sup>Tennessee, Montgomery, 284.
- <sup>27</sup>Tennessee, Wilson, 287.
- <sup>28</sup>Kentucky, Lincoln, 238.
- <sup>29</sup>Kentucky, Morgan, 239.
- <sup>30</sup>Indiana, Elkhart, 217.
- <sup>31</sup>Georgia, Camden, 203.
- <sup>32</sup>Wisconsin, Columbia, 302.

#### Winter Dysentery in Cattle

A winter dysentery has occurred in Canada for the past 20 years, usually in the fall coincidental with sudden drops in temperature, but also in the winter following extremely cold weather. It often appears simultaneously in all animals in the herd and with other herds in the area. Cattle 1 to 3 years old were most often affected. Before developing resistance, animals could be affected two or three times but never at less than a ten-month interval.

The first signs were a nasolacrimal discharge, diarrhea, and a mild temperature (104 F.) which soon dropped to normal or subnormal. The soft feces were often blood-flecked or hemorrhagic. The course was usually three to five days in the older, and seven to 14 days in the younger, animals, with a mortality of 1 to 2 per cent. Lesions were limited chiefly to the gastrointestinal tract, increasing from hyperemia of the abomasal mucosa to catarrhal inflammation with marked ecchymoses and swollen Peyer's patches in the jejunum and ileum, then receding to hyperemia from the cecum

posteriorly with petechiae in the posterior colon and rectum. The mesenteric lymph nodes were edematous.

The disease was transmitted to susceptible young cattle with fecal material given orally; also with a fecal filtrate (Seitz filter) given subcutaneously, thus indicating that it is a viral enteritis. The diphasic fever and leukopenia resemble those in bovine viral diarrheas (Indiana and New York). Infection is apparently maintained on premises from year to year, possibly in immune carriers or in a state of symbiosis until a stress factor precipitates the clinical syndrome. Two to four months after the viral infection disappears, a few animals may develop secondary bacterial infection with laminitis and erythema of the coronary band, as is found in mucosal disease.—*L. W. MacPherson in Canad. J. Comp. Med. (June, 1957): 184.*

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In a three-year study of winter dysentery, involving 25 dairy herds in New York, it seemed that infection had most often been introduced by an inseminator or other visitors, three to ten days previously. In two herds, new cattle may have introduced the disease; in two other herds, no source of infection was evident. There was no evidence of a dietary cause. An immunity was indicated, since a herd was seldom severely affected (80% of all animals) less than three or four years after its last attack.

Less severe outbreaks (10 to 50%) occurred at shorter intervals but the animals affected were usually those not present during the last attack. Cows 2 to 6 years old, recently fresh and lactating heavily, were most susceptible. Animals 4 to 24 months old were less severely affected and calves under 4 months seldom showed signs of infection. Temperatures were often high before signs appeared. Coughing was frequent and noticeable in 30 per cent of the herd, occasional in 40 per cent, and absent in 30 per cent. There was no evidence of Pasteurella infection or of benefit from vaccination.

Serum-neutralization tests showed this disease to be distinct from viral diarrhea. There was no leukopenia. Many vibrios were observed microscopically in the feces but pure cultures were not recovered. No treatment seemed effective.—*S. J. Roberts in Cornell Vet. (July, 1957): 372.*

# Rift Valley Fever as a Veterinary and Medical Problem

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ALTHOUGH the introduction of exotic diseases into the United States is always greatly feared, many of these diseases, even if introduced, would probably never spread in this country. This is particularly true when transmission depends upon special insect vectors which do not exist here; for example, trypanosomiasis (tsetse flies), leishmaniasis (*Phlebotomus* spp.), East Coast fever (ticks of the *Rhipicephalus* spp.), and heartwater (bont ticks). Where the specific vectors are present, as in Africa, these diseases constitute the major animal disease problems. Too great assurance can not be placed in these general observations, however, in view of the behavior of anaplasmosis in the United States. This disease is becoming an increasing problem and sometimes occurs in the absence of ticks. Its mode of transmission, apparently, has undergone modification.

There are a number of other exotic diseases which could spread with alarming ease in this country, either because their transmission does not depend on the presence of special vectors, or because vectors or other insects capable of transmitting certain diseases are present. Most of these are of a contagious type, like African swine fever, fowl plague, rinderpest, and foot-and-mouth disease. Authorities are constantly warning that *Aedes aegypti*, the vector of yellow fever, is prevalent in the southeastern states and that the introduction of the virus is all that is needed to spread the disease. The vectors of rabbit myxomatosis, are apparently present in the United States. In Africa, horsesickness and bluetongue of sheep are transmitted by the same vector. With bluetongue now enzootic in this country, conditions are apparently favorable for the spread of horsesickness should the virus be introduced. Another disease which could probably spread if the virus were introduced at a

favorable time and place is Rift Valley fever.

Rift Valley fever is an acute, infectious disease of sheep, cattle, and other animals, including man. Its importance to the animal industry lies in the high abortion rate which it produces in cattle and sheep, and the high mortality which it causes in lambs and calves. Although it causes a short febrile reaction in man, it is usually not fatal. The disease is caused by a small virus which is transmitted by a variety of mosquitoes and other insects but it is often contracted by man from contact, as in the performance of a necropsy.

## ETIOLOGY

The small virus ( $\pm 30 \mu$ ) is closely related to that of yellow fever. It can easily be adapted to embryonating eggs and has a host range much wider than most viruses, excepting perhaps rabies.

## HISTORY

Rift Valley fever derives its name from the Rift Valley of eastern Africa. It has occurred several times in Kenya, beginning in 1913, with heavy mortality in young lambs. In 1931, the virus was isolated, described, and recognized as the infective agent in some 200 human cases.<sup>1</sup> Although neutralizing antibodies have been found in serums of persons in a number of other central African localities, the virus has been isolated there only in Uganda, in 1944 to 1947.<sup>2</sup>

With only small populations of cattle and sheep in these areas, the active form of the disease has presumably not often been noticed, although it is probably enzootic there.

Up to 1951, there were only odd reports of the disease from central Africa and occasional reports of laboratory infections in England, the United States, and Japan.

Apart from Kenya, the disease was not regarded of economic importance until 1951 when it suddenly became widespread in the Union of South Africa. Sheep losses were estimated at 100,000 and human infections at 20,000.<sup>3-5</sup> The sheep losses were at first ascribed to bluetongue, enterotoxemia, vegetable poisoning, or internal parasites, and human cases were thought to be influenza. However, when a pedigree Holstein-Friesian bull suddenly died<sup>6</sup> and necropsy, by four veterinarians and two assistants, was followed within four or five days by illness in five of these workers, the cause was immediately investigated. The two diseases suspected were Rift Valley fever and Q fever. Today,

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the Wesselbron virus, the cause of abortions in sheep in South Africa in 1955,<sup>6,7</sup> would have to be included. As a result of this epizootic in South Africa, avianized live virus vaccine for sheep and cattle was developed.<sup>8</sup>

From the evidence available, the disease appears to be enzootic in an extensive area of the coastal plain of South Africa, and probably has been there for a long time.

#### EPIZOOTIOLOGY

In Kenya, the disease has appeared more or less explosively in sheep at intervals of five to seven years. It seems to be a jungle disease, living a cycle between insects and rodents, and appearing in domesticated areas more or less as an accident. When it appears, if there is a large population of susceptible animals and large numbers of vectors, an epizootic of a most explosive type is set off. The disease spreads with remarkable speed and usually, within a few weeks or months, the epizootic will have taken its course and disappeared. The morbidity is high.

Carriers of the disease are still being investigated; eight species of mosquitoes have definitely been associated with transmission.<sup>9</sup> It is highly probable that during an epizootic many kinds of blood-sucking insects act as mechanical vectors. If this virus were introduced into the United States in the summer or fall, it would probably spread rapidly. The type of country in South Africa, in which the disease occurs extensively, closely resembles the prairieland of this country.

In the absence of insect vectors, the disease usually does not spread in animals. Infected and susceptible sheep or mice can be together without danger of transmission from one to the other. While human beings are easily infected if they handle material rich in virus, spread from man to man by contact has never been encountered.

#### SIGNS AND LESIONS OF RIFT VALLEY FEVER

This disease has no pathognomonic clinical features. Its chief characteristics are an abrupt onset, short course, a sharp febrile reaction, and a death rate which is high in lambs and calves (up to 90%), but low in adult sheep (20%) and adult cattle (10%). Its most characteristic feature is a high abortion rate in cattle and sheep at all stages of pregnancy. In mice, the death rate from artificial infection is about 99 per cent. It is generally considered as non-

fatal for man but two deaths out of thousands of cases, have been ascribed to it. In cattle and sheep death is sudden, almost without warning; the rectal temperature, when taken, is high for a day or two prior to death. By comparison with other diseases, if material containing the specific virus is injected into sheep, the fever reaction is usually as follows: with Rift Valley fever and Wesselbron virus, the temperature usually rises on the afternoon of the second day, reaching a peak of 107 to 108 F. on the third day, then returning to normal within two or three days. The temperature reaction from Nairobi sheep disease virus is similar except that it may take about a day longer to rise. In bluetongue, the rise commences slowly on about the fifth day, reaching a peak about the seventh day, then dropping slowly; the typical clinical picture usually occurring after the peak of the fever. Heartwater, which is a rickettsial disease, has a much longer incubation period; the temperature usually begins to rise about the tenth day, takes about three days to reach its peak, remains elevated for a few days, and then recedes gradually. The death rate is high.

In its typical form, Rift Valley fever has an abrupt course, chronic cases being rare in all animals. The deaths usually occur soon after the temperature crisis, but abortions may occur for the next week or two. In man, the incubation period is slightly longer, usually four to five days. The disease is often mistaken for influenza because of its brief course and a similar febrile reaction with headaches, but the catarrh of the upper respiratory tract is absent in Rift Valley fever. Occasionally, there is hemorrhage into the retina causing temporary or permanent impairment of vision.<sup>10,11</sup>

**Pathological Changes.**—The most significant and consistent lesions are in the liver. They consist of tiny, circumscribed, grayish or dark foci disseminated throughout their substance. These lesions may coalesce, the entire liver becoming necrotic and sometimes yellow or light brown, due to icterus. However, the liver may retain its normal color; ordinarily, it is not enlarged.

The other lesions are less typical. They are found in many other acute febrile diseases and include general venous congestion and petechiae on the heart, in the lymph nodes, and in the alimentary tract.

The spleen may be slightly enlarged but it is often normal. Hepatitis probably occurs in man but, as the disease is rarely if ever fatal, the gross and microscopic changes in the liver have not been studied.

**Microscopic Changes.**—The principle lesion, essentially a circumscribed focal necrosis, is seen in the liver and is almost pathognomonic. Apparently, as the inflammatory reaction develops, histiocytes and neutrophils invade the lesion and become degenerated so that the entire lesion consists of degenerated liver cells and leukocytes. The size of the foci depends on the severity of the disease; in the most acute cases they coalesce and are extensive.

Acidophilic nuclear inclusions are also considered to be characteristic of Rift Valley fever. They are circumscribed and vary in size and number. These inclusions are most frequent in the livers of mice or young lambs and less so in the livers of adult sheep and cattle, particularly if these were killed or did not otherwise die a natural death.

#### DIAGNOSIS

Indicative of the disease are: (a) a mortality which is high in lambs and calves, much lower in adult sheep and cattle; (b) high abortion rates in cattle and sheep; (c) typical liver lesions; (d) infections in man, with short febrile course, after infective material has been handled. White Swiss mice are extremely susceptible and consistently die within two to four days after intraperitoneal injection. This is the only virus which, when administered by this route, will cause death in mice so quickly. Bacteria, like anthrax, erysipelas, and some clostridia may also do this but, if antibiotics are added to the inoculum, these are eliminated. A specific diagnosis can be made directly by dividing the inoculum into two parts and treating the respective portions with equal amounts of known negative and positive serums. As a rule, a diagnosis can be made within four days. The livers of dying mice are collected in 10 per cent formalin for histological examination. Simultaneous inoculation of sheep with the suspected material will also reveal the rather typical response.

#### SEROLOGY

In convalescent or recovered animals, antibodies can easily be demonstrated; they are present in abundance. The results of the serum-neutralization and comple-

ment-fixation tests are clear-cut. The antibody becomes demonstrable within a week after infection and persists for a long time; in man, it is still present after 20 years.<sup>12</sup>

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**Bats and Viruses.**—There is a strange compatibility between bats and viruses. Bats inoculated with the virus of Venezuelan equine encephalomyelitis or of Japanese B encephalitis build up a high concentration of the virus in three days and maintain it for up to 26 days before the virus apparently disappears. Since bats and mosquitoes live together, the former may be the disease reservoir and the latter the carrier to animals. During hibernation, the virus concentration in bats is lower but it is maintained for at least 90 days.—*Sci. News Letter* (July 13, 1957): 22.

# Surgery and Obstetrics

## and Problems of Breeding

### An Improved Hood for Swine Hysterectomies

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THERE HAS BEEN much interest in the past few years in the isolation of pigs at birth as a disease control principle. At birth, pigs are considered free from disease and, by careful handling and isolation, they can be kept free of many of the chronic swine diseases. By use of this principle, it has been possible to eliminate swine dysentery,<sup>1,2</sup> transmissible gastroenteritis,<sup>1,2</sup> atrophic rhinitis,<sup>3,4</sup> virus pneumonia,<sup>4</sup> and several unidentified enteric infections<sup>5</sup> from herds of swine.

To obtain disease-free pigs at natural birth is a tedious process, although this has been accomplished by a number of workers.<sup>3,6-10</sup> Due to the natural variations in gestation (111-118 days), it is difficult to predict when a gilt or sow will farrow. It is often impossible to have someone in attendance at all times. Furthermore, in farrowing, the interval between pigs may vary from ten minutes to one or more hours. This inconvenience is overcome by delivery of the pigs two to four days before expected parturition by hysterectomy,<sup>1</sup> cesarotomy,<sup>5</sup> or hysterotomy.<sup>11</sup> Using these techniques, there is less danger that the pigs will become contaminated.

The method of choice for obtaining disease-free pigs is by hysterectomy. The pigs are obtained conveniently with small risk of contamination. Since the dam is slaughtered and utilized for food, there is no hospital problem.

Based on experience obtained over several years, a portable, relatively inexpensive, and efficient hood has been developed for the convenient removal of pigs from the gravid uterus. This apparatus is described in this paper.

#### HOOD SPECIFICATIONS

The portable hysterectomy hood as shown in detailed drawings (fig. 1) can be constructed in most areas for less than \$200. It is made of galvanized iron and is 30 inches wide and 82 inches in over-all length. The interior floor is 30 by 48

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inches including the openings to the air lock tank (23)\* and the carrying case compartment (25). These openings are equipped with hinged covers to provide maximal space within the hood for the gravid uterus and pigs. The central section (24) is sloped for drainage and is covered by an elevated, removable, expanded metal working surface.

The top section, which is removable for cleaning, is sealed with sponge rubber weather stripping and is tightly secured with bolts and wing nuts. The hood temperature is maintained at 95 to 100 F. by a 2,000-watt, 115-volt calrod range surface unit (11) equipped with a three-heat switch (14) for manual temperature control.

Positive air pressure is maintained with a 6-inch squirrel cage blower (12). The air is drawn through a 6 by 24-inch No. 50 FG spunglass filter-down pad (15),† passed over the heating unit, and into the hood. A 20-watt fluorescent lamp (9) over a plexiglass window (19) lights the interior. For better coverage of the working area, the plexiglass windows (19) and the armholes (22) are not centered. On the right side, the operator is placed nearer the tank and on the left side nearer the blower. The armholes have medium-weight canvas sleeves (21) held in position by adjustable metal bands (20). A dairy-type scale (5) with a hook extended through a light rubber diaphragm into the hood makes it possible to weigh the pigs during the operation. The pigs are weighed in a small canvas bag.

The air lock tank has a 6-inch baffle (2) to provide expansion space for the liquid and still maintain the air lock when the uterus is removed. The door (17) on the carrying case compartment has a weather-stripping seal and is kept closed with a trunk hasp. The unit is equipped with 4-inch, hard rubber casters.

The carrying cases for pigs (26 in. long, 13 in. wide, and 8 in. deep) are made of ½-inch plywood and have canvas covers with 15-inch zippered openings. These boxes are kept in oversized canvas bags for sterilizing and transporting.

#### USE OF HOOD

In preparation for a hysterectomy, the hood is sterilized with formaldehyde gas the day preceding the operation, using the following procedure: (1) the tank is filled to about 4 inches from the top with a mild antiseptic solution also filling the drain trap; (2) the sleeves which have been steam-sterilized are attached; (3) the filter is checked for leaks but need not be

\*The figures in parentheses refer to parts of the hood as illustrated in figure 1.

†American Air Filter Co., Louisville, Ky.

Fig. 1—Detailed scaled drawings of the hysterectomy hood: (1) air lock tank and drain valve; (2) baffle; (3) liquid level; (4) shelf to guide uterus into tank; (5) scale; (6) shelf for inside equipment; (7) hinged lid covering tank; (8) hinged lid covering carrying box opening; (9) light; (10) guard covering heating unit; (11) electric heating unit; (12) blower; (13) switch-blower; (14) switch-heating unit; (15) air filter, frame, and trunk hasp lock; (16) power supply; (17) door carrying case compartment; (18) drain floor and drain; (19) plexiglass windows; (20) ring for holding sleeves; (21) canvas sleeves; (22) armhole openings; (23) air lock tank inside and outside openings; (24) drain and expanded metal cover; and (25) carrying case compartment and opening.

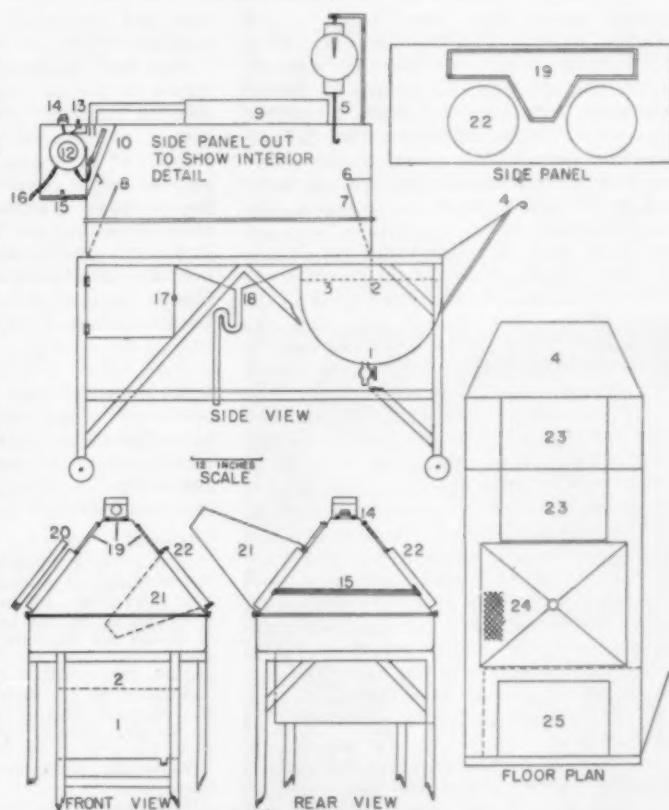


Fig. 2—Photograph of the hood during a hysterectomy.

changed more than every three to six months, depending on the accumulation of dirt; (4) the hood is covered with a plastic drape; and (5) a pan containing 35 Gm. of potassium permanganate, to which has just been added 150 ml. of 40 per cent formaldehyde is immediately placed in the carrying case compartment. The formaldehyde pan is removed in about ten minutes. The carrying cases within the canvas bags are sterilized with formaldehyde gas, using the same procedure and about one fifth the amount of chemicals. One hour prior to the hysterectomy, the blower fan and the heater are turned on to bring the hood to operating temperature and remove excess formaldehyde gas.

For the hysterectomy (fig. 2), the two hood operators and the surgeon thoroughly scrub their hands and arms with soap, water, and a mild antiseptic solution. A previously sterilized package containing towels, weighing sack, umbilical clips (small, electrical, alligator-type), string, and scissors is passed into the hood through the carrying case door. The package is double-wrapped in paper with the outer wrapping being removed before passing the bundle into the hood. The carrying case is slipped from the canvas bag into the compartment with a minimum of exposure.

The sow is hoisted by the hindlegs into operating position and the belly area is scrubbed with water and antiseptic solution. The animal is then lowered into a 55 gal. barrel filled with  $\text{CO}_2$  gas. The barrel is easily filled with  $\text{CO}_2$  gas by pulverizing about 3 lb. of dry ice in the barrel a few minutes before being needed. After the sow has inhaled the  $\text{CO}_2$  gas for one minute, the barrel is removed and an aseptic abdominal incision is made.

The hysterectomy hood is rolled into position so that the upper edge of the tank shelf (4) meets the lower end of the incision. The gravid uterus is rolled out onto the shelf, the cervix is severed, and the uterus slides down into the tank (1). The hood operators quickly pull the uterus out of the tank and into the hood, close the tank door, and speedily tear the pigs free from the uterus. This operation should take less than one minute. Clips are placed on the umbilical cords which are then severed. The pigs are wiped dry with the sterile towels. The umbilical cords are then

tied and cauterized with an iodine-alum mixture.

Pigs are weighed, marked, and transferred to the carrying case. The carrying case is removed from the hood and returned to the canvas bag, limiting the amount of exposure, as before. The uterus and the soiled towels are removed through the carrying case door and the hysterectomy procedure can be repeated. The antiseptic solution in the tank can be changed between hysterectomies providing the blower fan is kept in operation to maintain positive pressure within the hood.

#### SUMMARY

An improved hysterectomy hood for two operators is described. The unit is portable, efficient, and can be built for approximately \$200. It can be used anywhere electricity is available. The procedures used are described in detail.

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## Achalasia of the Esophagus with Megaesophagus in a Dog

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Since 1679, when the first case of esophageal obstruction was described and treated in man,<sup>4</sup> many terms have been applied to this condition, such as phrenospasm, simple ectasia, idiopathic dilatation, and dystonia.<sup>7</sup> Cardiospasm, the term now most widely used, seems incorrect since the cardia of the stomach is not involved. Therefore, the term achalasia seems more acceptable and will be used.

Achalasia<sup>3</sup> results from a neuromuscular imbalance characterized by dysfunction of the esophageal wall and the failure of the terminal sphincter of the esophagus to relax after swallowing.<sup>1,12</sup> The result is an accumulation of ingested material and saliva with dilatation or megaesophagus proximal to the sphincter. There is persistent emesis.<sup>12</sup> In the dog, achalasia is uncommon but not rare.<sup>6</sup>

Absence of ganglion cells of the myenteric plexus appears to result in an imbalance between the parasympathetic and sympathetic innervation of the esophagus.<sup>2,7,10,12</sup> This becomes manifest shortly after birth.

### CASE REPORT

A spayed German Shepherd, 8 months old, had experienced intermittent emesis since 6 weeks of age. She was fed daily at 9 a.m., noon, and 4 p.m. The vomiting usually occurred about twice daily between 10 p.m. and 6 a.m. The vomitus consisted of solid, undigested food, mixed with saliva. Various feeding schedules and treatments had failed to improve the condition. She was cachectic, weighed only 34 lb., and was irritable, but otherwise seemed normal.

The dog was anesthetized with pentobarbital sodium, and esophagoscopy revealed a markedly inflamed esophagus with the thoracic portion extremely dilated.

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The esophagus was then filled with 500 ml. of barium solution (30 to 50%) and a lateral radiograph was taken (fig. 1). It showed a dilated cone-shaped esophagus with a terminal constriction.

The following day, the first bougienage (dilation with mercury-filled bougies) of the esophagocardial orifice was performed. The technique consisted of tying the anesthetized dog on an operating table, tilting the table top vertically with the dog's head



—Med. Illust. Lab., Syracuse V.A. Hospital, Syracuse, N. Y.  
Fig. 1—Radiograph showing the barium-filled esophagus of a dog. Note distal constriction.

up, and introducing the bougies in series through the esophagus into the stomach. Tubes of successively larger diameter were passed and each held in the stenotic orifice for approximately three to five minutes until the orifice was sufficiently dilated.<sup>2,12</sup> A radiograph was taken after the first bougie was passed, to determine its position. The procedure was repeated in two weeks. All vomiting disappeared shortly after the second treatment. A year later, the dog was in excellent physical condition

and her weight had increased from 34 to 52 lb.

#### DISCUSSION

Even with the best techniques, the results of mechanical dilatation are not wholly successful, so surgical intervention is sometimes a necessity. However, mechanical dilatation has given satisfactory results;<sup>1</sup> the Mayo clinic, in 1951, reported that 81 per cent of dilatations with bougienage<sup>2</sup> were satisfactory. Success was reported in a dog with mechanical dilatation along with a bland diet and atropine.<sup>3</sup>

The procedure does not restore esophageal peristalsis or tone but it seems to modify the contractile power of the sphincter, permitting the esophagus to empty which, in turn, allows recovery of the muscular tone of the esophagus.<sup>5,12</sup>

When the human patient fails to respond to conservative treatment, surgical intervention, such as Heller's extramucosal myotomy, is employed.<sup>4</sup> Recently, an excellent article appeared advocating surgery for achalasia in dogs.<sup>6</sup>

Bougienage is not difficult and bougies can be obtained through a human hospital.

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*Abortion in Swine Due to Excessive Nitrate.*—Sows that were well cared for were

pastured on oats and rape in a lot which had been previously used as a feedlot for cattle. Some aborted about two weeks before full term while the pigs of others were dead or weak at birth and the pigs of some had physical abnormalities. Serological tests were all negative for leptospirosis and brucellosis, but other tests indicated a low level of serum vitamin A and the presence of excessive nitrate. Tests showed that the nitrate content of the rape was 5.52 per cent and of the oat plants 0.53 per cent—a total high enough to produce vitamin A deficiency and too high for a safe ration even for swine. If cattle or sheep had been grazed on this pasture for any length of time, they probably would have had acute nitrate intoxication.—A. A. Case in Sheep Breeder (July, 1957): 12.

*Laparoecectomy in Horses.*—Surgery was performed on 3 horses, in Poland, under chloral anesthesia, for removal of material impacted in the cecum. All recovered. A 20-cm. (8-in.) incision was made on the right side of the abdomen parallel to and 15 cm. from the costal arch. A 20-cm. incision was then made along the dorsal band of the exposed tip of the cecum. Upon completion of the operation, antibiotic therapy was recommended but not considered indispensable.—Med. Weteryn. (Jan., 1957): 29.

*A New Lethal Gene in Cattle.*—A lethal gene which prevents the development of the pituitary gland in the bovine fetus, eventually causing death, has been reported by veterinarians at the University of California. To date, it is known to affect only one line of Guernsey cattle. The fetus ceases to grow at 7 months but birth is delayed until after 292 to 526 days of gestation; an average of 401 days. Calves delivered by cesareotomy lived only a few minutes. This condition differs from hereditary-prolonged gestation.—Brown Swiss Bull. (Aug., 1957): 75.

*Effect of Freezing on Vibrio Fetus in Semen.*—When bull semen was inoculated with millions of *V. fetus* organisms per milliliter without the addition of antibiotics, the number of organisms was decreased by over 99 per cent during the process of freezing.—W. N. Plastridge in A. I. Digest (July, 1957): 17.

# Clinical Data

## The Action of Chlorpromazine Hydrochloride in Calves

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IN HUMAN MEDICINE, one of the most useful properties of the relatively new tranquilizing drugs is their ability to relieve anxiety without producing other undesirable effects of sedatives. Patients under treatment remain fully conscious but are so relieved of their mental depressions that they have a feeling of well being.<sup>6</sup> The action of these drugs has also been described in dogs,<sup>2,3,7</sup> horses,<sup>6,7</sup> and cattle.<sup>1,7</sup> They are being used routinely in managing vicious and nervous dogs.

There are numerous situations where drugs with tranquilizing action would be most useful if successfully applied to cattle. The trying period following weaning would, perhaps, be less of a shock to calves if anxiety could be removed. This post-weaning period also provides a test situation for the study of the psychological effect of tranquilizing drugs in cattle.

Beef calves which have not been handled previously are commonly removed from their native pastures, weaned, shipped, and placed in strange feedlots all within a few days. It is thought that the stress set up by these procedures results in physiological reactions which lower the resistance of the animals sufficiently to favor the development of the shipping fever complex. A similar disease syndrome is commonly seen following the weaning of calves which will remain on the ranch. In this case, factors such as shipment and exposure to abnormal physical elements are not involved. Change of feed in such ranch calves seems to be of minor importance since most of the beef-type cows on dry western ranges give little milk at this time of the year. This is borne out by the fact that these calves gain little weight during the month before weaning.

Whether anxiety and apprehension contribute sufficiently to "stress" to cause physiological dysfunctions is not known.

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These calves do a great deal of "fence walking" and bawling for several days after weaning. They often refuse feed and water to the point that they actually lose weight for a week or two.

Chlorpromazine\* (10- $\gamma$ -dimethylaminopropyl)-2-chlorophenothiazine hydrochloride) is one of the tranquilizing drugs which has been tested in animals. For use in cattle, it has the advantage of being in solution for parenteral administration. The following report deals with observations on the use of chlorpromazine hydrochloride in calves in an effort to relieve the anxiety associated with weaning.

### MATERIALS AND METHODS

The beef calves used were provided through the cooperation of the Animal Husbandry Department, University of Nebraska, and the Beef Cattle Research Station at Fort Robinson, Neb. They were approximately 6 months old and had been on pasture with the cows. While most of the calves were Herefords, a few Angus and Shorthorns were also studied.

The chlorpromazine hydrochloride, supplied in 1- and 2-ml. glass ampules (containing 25 mg./ml.), was injected intramuscularly in the cervical region. The first experiment was conducted to determine the single dose which would be effective for the longest time without producing excessive depression or toxic reaction.

Clinical observations following injection were supplemented by biochemical blood analyses, blood cell counts, and weight-gain determinations.

### EXPERIMENTAL PROCEDURE AND RESULTS

The initial trials to determine drug tolerance and dosage were made on 3 dairy calves 6 to 8 months old which weighed about 400 lb. These calves had been weaned at about 1 month of age and were docile. They had been handled frequently for the taking of blood samples for other experiments.

*Experiment 1.*—The first calf was given the recommended dose of the drug, 1.5 mg. per kilogram of body weight.<sup>7</sup> Twenty minutes later, it was highly excited, probably fighting the halter to which it was not accustomed. A half hour after in-

\*The drug (Thorazine) used in these trials was furnished by Smith, Kline, and French Laboratories of Philadelphia, Pa.

jection, when freed in its own pen, the calf was quiet but showed incoordination of the hindlegs which soon became so pronounced that it could hardly stand. This unsteadiness was severe for six hours, then decreased, but it was evident for two more days. When not disturbed, the calf would lie down and draw its head around toward its flank, a position later noticed to be characteristic of treated calves. It would get up readily and seemed normally sensitive to the pain induced by the bleeding needle. The nictitating membranes were noticeably prominent.

The calf was hungry when fed two hours after the injection. Rumen activity was absent at one hour postinjection but was normal two hours later. Regurgitation was normal in spite of the fact that chlorpromazine hydrochloride has a powerful anti-emetic action in dogs and man.<sup>4</sup> The heart rate increased from a preinjection 100 per minute to a postinjection 160 at four and seven hours, but was normal at 24 hours. The respiratory rate was increased somewhat at one hour, but normal at five hours. The results of blood studies after injection are shown (table 1). This calf ate very little for four days following injection.

This dosage seemed excessive; therefore, 2 similar calves were injected with a smaller amount of the drug (1.25 mg./kg. of body weight). These and an untreated control calf were kept under observation and subjected to blood studies. The treated calves showed only a mild reaction to the drug. They acted tired, preferring to lie down when not disturbed. After regurgitation, they would sometimes forget to chew the cud. Mild incoordination was present

and they scarcely resisted being bled. Rumination was normal and both calves ate and drank normally. Both experienced tachycardia, the heart rate being 140 per minute at one and one half hours. The various chemical constituents of the blood remained constant. The hematocrit values decreased and the erythrocyte counts dropped from 10.2 million to 8.5 million at two and one half hours and five and one half hours, but both were normal at 20 hours. Leukocyte counts did not change appreciably.

**Experiment 2.**—The second trial was made on purebred beef calves which were raised on pasture. They were about 6 months old and weighed 250 to 450 lb. On the basis of weights taken a week before weaning, 10 of the bull calves were injected with chlorpromazine hydrochloride (1.25 mg./kg. of body weight); 21 bull calves and 43 heifers were not injected.

All of the injected calves developed some degree of incoordination of the hindlegs. One was staggering in less than five minutes. Fifteen minutes after injection, another could hardly stand and 7 of the 10 were lying down; all of the untreated calves were up and anxious to get back to the cows. Some of those lying down had their heads characteristically drawn back toward their flanks. The treated calves looked sleepy, but got up and down of their own accord, walked unsteadily, ate weeds along the fence, and ruminated normally.

About one and one half hours after the time of injection, the calves, treated and untreated, were loaded into trucks and hauled 14 miles to the campus feedlots. Several of those treated were unsteady and

TABLE I.—Physiological Observations on a Calf Injected with Chlorpromazine Hydrochloride at the Rate of 1.5 mg./kg. of Body Weight. This Calf Showed Signs of Intoxication

	Pre-injection	Hours postinjection				
		1/2	1 1/2	2 1/2	5	7
Erythrocytes (millions/cm.)	10.7	10.5	8.9	9.9	9.5	9.9
Hematocrit (%)	41.0	42.0	38.4	36.2	36.2	37.8
Leukocytes (thousands/cm.)	13.5	13.5	15.5	16.2	20.4	16.3
Neutrophils (thousands/cm.)	3.2	3.7	4.0	5.2	9.0	8.6
Lymphocytes (thousands/cm.)	10.2	9.5	11.1	11.1	11.4	7.7
Plasma glucose (mg./100 ml.)	67.0	75.0	95.0	105.0	103.0	98.0
Plasma nonprotein nitrogen (mg./100 ml.)	21.8	30.0	21.8	21.8	21.8	19.6
Plasma magnesium (mg./100 ml.)	2.84	2.84	2.73	2.73	3.07	3.07
Whole blood magnesium (mg./100 ml.)	2.66	2.55	2.55	2.55	2.84	2.84
Plasma phosphorous (mg./100 ml.)	8.40	7.42	7.75	8.00	9.49	8.15
Plasma calcium (mg./100 ml.)	12.10	11.71	11.20	10.85	11.50	11.30
Erythrocyte cholinesterase ( $\mu$ M/ml./hr.)	164.0	164.0	154.0	164.0	156.0	150.0
Body temperature (F.)	102.6	102.8	102.0	.....	101.8	.....
Heart rate/min.	100.0	160.0	136.0	144.0	160.0	160.0
Respiratory rate/min.	36.0	50.0	34.0	.....	32.0	32.0

difficult to load, but none had to be carried up the chute. On arrival, 3 of the calves were lying down in the truck and 1 had to be helped up.

After unloading, 1 of the treated calves immediately started to eat silage in a feed bunk and soon 2 additional treated calves were eating at another bunk. In the meantime, the other calves were investigating the fences and bawling. Within ten minutes, about 30 of the untreated calves were at the feed bunks, possibly following the example of the quieter treated calves, several of which were eating. After arrival in the feedlots, the bull calves were penned separately from the heifers.

The treated calves did almost no bawling; in fact, all of the bull calves (treated and untreated) were much quieter than the heifers and also did much less fence walking. The behavior of the treated calves appeared to have a quieting effect on their untreated penmates. The difference in behavior between the bull calves and the heifers could be noticed for three days. The incoordination of the hindlegs disappeared entirely by the end of the second day and no particular tendency to lie down was noticed in the treated calves after the first day.

The average weight of the treated calves was approximately 20 lb. less than the controls. During the week previous to weaning and treatment, the average gain of the treated calves had been 1.8 lb. while that of the untreated bull calves had been 4.7 lb. During the week following weaning, the average gain of the treated calves was 15.6 lb. while that of the untreated controls was 13.9 lb.

Since calves lying down would be injured in transit, the dosage of chlorpromazine used in this experiment was still considered too large for use before shipment.

**Experiment 3.**—The third trial was made on beef calves raised under western ranch conditions. They were more nervous than those used previously. Thirty-five calves were picked at random, representing all of the weight groupings, and were injected with chlorpromazine hydrochloride at the rate of 1.0 mg./kg. of body weight.

Incoordination of the hindlegs was again noticed, but was not as severe as in the previous trials. Many of these calves lay quietly in the holding pens and none were bawling. They did not lie down in the

trucks when hauled 7 miles to the winter feedlots. On arrival, they immediately ate and drank and soon were lying down. The untreated control calves in the adjoining pens bawled constantly and appeared excited.

The next morning, the two groups of calves continued to show the same attitudes. The untreated calves were walking around the fences and bawling and at least half of them were gaunt. The treated calves were eating and drinking normally and seemed contented. About 30 hours after the calves were weaned and injected, several loads of culled cows were brought into holding pens about a half mile from the calves. This markedly disturbed the untreated calves; at least 90 per cent of them bawling more than previously and milling along the fence, at times almost running. The injected calves did not appear to be greatly disturbed, either by the cows or by the confusion created by the untreated calves in the adjoining pen.

Forty-four hours after injection, the treated calves were quietly eating, with an occasional low bawl being heard. The untreated calves were still highly excited.

When weighed again six days after weaning, the average loss for the 35 treated calves was 6.0 lb. compared with 6.9 lb. for the 174 untreated calves.

A respiratory disease, clinically similar to shipping fever, commonly occurs after the weaning of calves which remain on the home ranch. Only 1 of the 35 chlorpromazine-treated calves (2.9%) became sick enough to require antibiotic treatment for respiratory disease, while 13 (7.5%) of the 174 control calves required treatment.

**Experiment 4.**—In the fourth trial, 20 calves which were to be shipped 250 miles from a ranch in western Nebraska to the University feedlots in Lincoln were injected with chlorpromazine hydrochloride (1.0 mg./kg.) after being weaned and 17 hours before being loaded into trucks.

After injection, 1 calf showed considerable incoordination; the others were quiet and ate or lay down, while the action of the untreated group was typical of newly weaned calves.

The next morning, just before loading for the five- to six-hour trip, and again after being unloaded, blood samples were taken from 5 of the treated and 5 of the untreated calves. None of the calves lay

down in the trucks and no incoordination was noticeable on arrival (23 hours after injection). Chemical analyses of the blood samples are shown (table 2). Blood counts

TABLE 2—Mean Blood Values of 10 Calves Before and After a Six-Hour Shipment by truck

	Treated with chlorpromazine 1.0 mg./kg. body wt.		Untreated	
	Pre- shipment	After shipment	Pre- shipment	After shipment
Hematocrit (%)	43.0	39.9	45.2	43.5
Hemoglobin (Gm./100 ml.)	15.8	14.3	15.3	14.1
Whole blood ascorbic acid (mg./100 ml.)	0.25	0.27	0.35	0.31
Plasma chlorides (mEq/liter)	98.0	92.0	96.0	95.0
Erythrocyte cholinesterase ( $\mu$ M/ml./hr.)	84.3	97.5	98.4	104.4
Nonprotein nitrogen (mg./100 ml.)	24.8	23.8	25.9	23.5

made on the heparinized blood samples taken before shipment (6 hours before counting) were unsatisfactory.

The temperatures of the calves were taken daily after arrival in the feedlots. Of the 20 treated calves, 2 (10%) had temperatures over 104 F. during an 11-day period, compared with 12 (31.6%) in the group of 38 untreated calves. The significance of these high temperatures is open to speculation.<sup>5</sup> No animal from either group developed signs of shipping fever.

The 20 injected calves gained an average of 25.8 lb. in the first week after shipment compared with 19.2 lb. for the 38 control calves.

#### DISCUSSION

While these investigations are preliminary in nature, some tentative conclusions may be drawn. For calves approximately 6 months old and weighing 300 to 400 lb., 1.5 mg. of chlorpromazine hydrochloride per kilogram of body weight caused an excessive amount of incoordination and produced signs of intoxication. A dosage of 1.25 mg./kg. was tolerated well by dairy calves, but caused incoordination in the beef calves. When 1.0 mg./kg. was used, incoordination was present but was not objectionable.

Chlorpromazine hydrochloride, in non-toxic doses, apparently greatly reduced the anxiety associated with weaning and, except for a tendency to lie down, the calves did not have the appearance of animals un-

der the effects of sedatives. They were alert enough to drink and eat normally, seemed entirely conscious of their surroundings, but reacted less violently to the unfamiliar stimuli of handling, confusion, and weaning. In trial 3, the effects of the drug were still obvious 44 hours after injection.

The treated calves made better gains than the controls during the week following weaning. It is possible that a longer period of drug action would have accounted for a greater weight gain advantage. Attempts should be made to prolong the drug action by using repeated injections or a repository base to decrease the rate of absorption. Since no cases of shipping fever developed in the control calves, the importance of anxiety as a predisposing stress factor could not be evaluated.

#### SUMMARY

Newly weaned beef calves treated with chlorpromazine hydrochloride were much less disturbed by the shock of weaning than untreated control calves. In three trials, the average weight gain during the week following weaning was greater in the treated than in untreated calves. A single dose (1 mg./kg. of body weight) of the drug produced tranquilizing action without signs of intoxication for up to 44 hours.

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## Field Studies with Piperazine-Carbon Disulfide Complex Against Parasites of the Horse

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RECENT REPORTS<sup>1-3</sup> indicate a broad spectrum of activity by the piperazine-carbon disulfide complex\* against the common internal parasites of the horse. In addition to the high level of efficacy against the ascarid and small strongyles, there was a significant removal of bots, one species of large strongyle (*Strongylus vulgaris*), and mature pinworms. The present study was undertaken to extend the evaluation of this compound under field conditions.

### MATERIALS AND METHODS

All age groups, sucklings, weanlings, yearlings, and mares, from several horse farms in the central Kentucky region were included in the study. The body weight of each animal was estimated, and a suspension\* of the drug was administered via stomach tube at the rate of 0.5 oz./100 lb. of body weight. This dose rate delivered approximately 37.5 mg. of the drug per pound of body weight (20 mg. of piperazine base per lb., and 4.5 drams of carbon disulfide per 1,000 lb. of body weight). Groups M6, M7, and M8 were given smaller doses, 0.33 oz./100 lb. or 25 mg./lb. No pretreatment fasting was practiced other than to withhold the grain ration on the morning of treatment.

The efficacy of treatment was judged from pre- and post-treatment worm egg and larval counts on fecal samples. The methods used for these counts were described earlier.<sup>2</sup> Only one pretreatment count was made, whereas several post-treatment examinations were made at intervals over periods as long as 15 weeks.

### RESULTS

The eggs per gram (e.p.g.) observations are presented as averages for each group (table 1).

Only one of the 17 groups of young animals did not have ascarid eggs before treatment, and ascarid e.p.g.'s made one

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\*Parvez (Upjohn Co.) suspension of the piperazine carbon disulfide complex, 250 mg./cc.

week after treatment were consistently negative except for 1 animal in each of groups M3, M6, and M7, and 3 animals in group M5. The reduction of ascarid e.p.g. for these 6 animals averaged 91 per cent. Of special interest was the failure of ascarid eggs to reappear in the feces until the tenth week after treatment.

Striking reduction of strongyle e.p.g. was a characteristic finding in the examinations made one week after treatment. More complete removal of strongyle eggs was observed in yearling groups W1 and W2 that were given 18.75 Gm. of phenothiazine along with the dose of piperazine-carbon disulfide complex. Tendency for strongyle e.p.g. to increase was not noticed until the fifth week post-treatment. Generally, lower strongyle egg counts, before and after treatment, were associated with the low-level phenothiazine medication in groups with the "M" designation.

The larvae per gram (l.p.g.) counts are presented as averages for each group (table 2). The larvae of one large strongyle, *S. vulgaris*, were differentiated from all other strongyle larvae ("str"), most of which were small strongyles.

In general, striking reductions in "str" larvae followed treatment, corresponding with the drop in strongyle e.p.g. Furthermore, the number of these larvae did not increase until the fifth week which is in accord with the egg counts. Comparatively small numbers of larvae were recovered from cultures prepared from animals that were on low-level phenothiazine treatment.

In contrast with the foregoing, the number of *S. vulgaris* larvae was not reduced by treatment. This was particularly evident in groups L3 and L4. Larvae of this species were not present in the pretreatment cultures prepared from the weanlings and did not appear until several months later. Practically no *S. vulgaris* larvae were found in the animals that were on low-level phenothiazine treatment.

Infective larvae of *Strongyloides westeri* were generally present in the cultures prepared from the younger animals. Counts of

TABLE I—Egg Count Data on the Efficacy of Piperazine-Carbon Disulfide Complex Against Ascarids and Strongyles of the Horse

Animals	Pre-treatment	Eggs per gram feces — group average Post-treatment (week)											
		1	2	3	4	5	6	8	10	12	13	15	
Group Age No.	A	S	A	S	A	S	A	S	A	S	A	S	
C1 N 7	525	545	B	15	—	—	—	—	—	—	—	—	
E1 W 8	310	800	0	5	0	1	0	10	0	25	0	110	
L1 W 6	10	230	0	5	—	—	—	—	0	655	—	—	
L2 W 14	165	340	B	10	—	—	—	—	—	—	0	1,480	
M1* W 12	0	40	0	2	—	—	0	2	—	—	0	70	
M2* W 10	10	25	0	0	—	0	0	0	5	—	0	15	
L3 Y 6	260	1,040	0	45	—	—	0	340	—	—	35	320*	
L4 Y 14	80	1,480	B	50	—	—	0	400	—	—	65	195*	
M3* Y 11	150	105	1	5	—	—	0	5	—	4	190	20	
M4* Y 10	145	125	0	5	—	—	0	3	—	0	85	235	
M5* Y 20	150	265	8	10	—	—	8	8	—	4	55	10	
E2 Y 5	225	230	0	10	—	0	0	—	—	0	235	—	
W1* Y 16	55	155	0	1	—	—	—	—	—	—	—	—	
W2* Y 14	35	135	0	1	—	—	—	—	—	—	—	—	
M6* Y 10	20	270	4	4	—	—	—	—	—	—	—	—	
M7* Y 6	335	215	1	4	—	—	—	—	—	—	—	—	
M8* Y 15	10	115	0	4	—	—	—	—	—	—	—	—	
C2 M 6	0	1,040	0	15	—	—	—	—	—	—	—	—	
E3 M 4	0	1,165	0	160	0	245	0	95	0	195	0	210	
J1 M 3	0	1,330	0	10	—	—	—	—	0	585	—	0	

\*=18.75 Gm. phenothiazine included in dose; \*on low-level phenothiazine; N=suckling; W=weanling;  
Y=yearling; M=mare; A=ascarid; S=strongyle.

these larvae were not made; however, gross estimates of the numbers present before and after treatment gave no indication that the drug was active against this species.

Several animals in groups M3, M4, and M5 showed an impairment of appetite and voided feces of softened, mushy consistency during the day following treatment, the only untoward effects ascribable to the treatment.

#### DISCUSSION

The highly effective removal of ascarids by the piperazine-carbon disulfide complex at the dose rate of 0.5 oz. of the suspension

per 100 lb. of body weight in these observations is in agreement with previous critical tests<sup>1</sup> with this compound. Limited evidence in the present tests suggests that a still lower dose (0.33 oz./100 lb.) is equally effective in removing mature ascarids. Of special interest was the failure of ascarid eggs to reappear until the tenth week post-treatment, which corresponds with the normal period of development required by the ascarid in the horse. This indicates the effective removal of the immature ascarids in the gut lumen at the time of treatment and offers promise of a longer interval between treatments than has been recom-

TABLE 2—Larval Count Data on Efficacy of Piperazine-Carbon Disulfide Complex Against Strongyles of the Horse

Animals	Pre-treatment	Larvae per gram feces — group average Post-treatment (week)											
		1	3	4	5	6	8	10	12	13	15		
Group	Age	No.	S.v.	Str.	S.v.								
L1 W 3	0	180	0	1	—	—	—	—	5	125	—	7	94
L2 W 9	0	200	0.1	2	—	—	—	—	17	205	17	326	23
M1* W 12	0	2	0	0	—	0	1	—	0	10	0.1	24	.07
M2* W 10	0	4	0	0	0	0.2	0	0.1	—	0	5	0	18
L3 Y 3	7	94	18	12	—	—	28	65	—	0*	3*	—	—
L4 Y 8	23	252	28	14	—	—	37	74	—	0	38	0.2*	26*
M3* Y 11	.07	32	0.02	1	—	0.02	4	—	—	0	18	—	—
M4* Y 10	0	18	0	0	—	0	1	—	—	0	18	—	—
E2 Y 3	3	175	2	19	3	2	—	—	6	42	—	—	—
M6* Y 10	0	29	0	2	—	—	—	—	—	—	—	—	—
M7* Y 7	0	37	0	1	—	—	—	—	—	—	—	—	—
J1 M 3	0	380	0.3	4	—	—	—	—	—	—	—	—	—

\*On low-level phenothiazine; W=weanling; Y=yearling; M=mare; S.v.=Strongylus vulgaris; Str.=all other strongyles.

mended<sup>4</sup> with carbon disulfide. Additional trials designed to provide data on the feasibility of an eight-week interval between treatments with piperazines for ascarid control are underway and will be reported later.

Marked reduction of strongyle e.p.g. following treatment with piperazine-carbon disulfide complex is in accord with earlier trials<sup>1</sup> which indicated that a similar (34 mg./lb.) dose level would effectively remove small strongyles. Larval count (l.p.g.) data were generally in agreement except for the larvae of the most pathogenic strongyle, *S. vulgaris*, which were not reduced by treatment. The partial removal (34 to 65%) of this species reported<sup>1</sup> from critical tests was found at higher dose levels (45.5 to 91 mg./lb.). The critical tests and the present observations indicate that the 37.5 mg./lb. dose should not be relied on for the control of *S. vulgaris*.

The effective functioning of low-level phenothiazine medication in strongyle control was also evident in these trials. Depression of egg production and inhibition of larval development, particularly *S. vulgaris*, were associated with animals on this treatment.

#### SUMMARY

Treatment of 197 sucklings, weanlings, yearlings, and mares with piperazine-carbon disulfide complex at 37.5 mg. per pound of body weight (0.5 oz. of suspension/100 lb.) under field conditions resulted in the effective removal of both immature and mature ascarids, and small strongyles. This dose level showed no apparent action against *Strongylus vulgaris* and *Strongyloides westeri*.

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*Lead Poisoning.*—Three persons in one family developed cramps, vomiting, a metallic taste, and other signs of lead poisoning within two hours after eating a duck which had been shot, then stored in a freezer for two months. Chemical analysis showed 0.12 per cent lead contained in the meat.—*Pub. Health Rep.*, April 27, 1957.

*Vacuolated Neurons in Healthy Sheep.*—Vacuolation of the neurons of the medulla has been considered an essential lesion of scrapie. They were always present but often smaller in experimentally produced cases. The recent finding of a high incidence of similar vacuoles in 39 healthy sheep, mostly Cheviots, suggests that they are either in the incubative stage of scrapie, have a nonclinical form of scrapie, or that vacuolation is not directly connected with scrapie.—*Zlotnik and Rennie in J. Comp. Path.* (Jan., 1957): 30.

*Ingestion of Broken Glass.*—Swallowed broken glass seldom causes illness. Neither toxic effects nor lesions in the gastrointestinal tract occurred when ground glass or various-sized particles of glass were fed to dogs (in 1918). If the patient is seen soon after the ingestion of glass, large quantities of mucilaginous substance should be given and then vomiting induced. After this, large amounts of mucilaginous or soft bulky indigestible material should be given. If bowel movements seem retarded, an enema of mucilaginous or oily material is indicated. Signs of perforation indicate prompt surgical intervention.—*J.A.M.A.* (March 16, 1957): 999.

*Tick Transmission of Disease.*—Ticks can be responsible for diseases in livestock by transmitting "toxins" as well as viruses, piroplasms, anaplasms, rickettsias, and *Borrelia* organisms. Transovarian transmission has been established in some ticks for many of these infective agents; therefore, ticks act as reservoirs as well as

vectors of disease. Of two toxin diseases, tick paralysis and "sweating sickness," the former, caused by a neurotoxin, has been reported in man, domestic animals, and birds, while the latter, which occurs in Central, East, and South Africa, seems to affect only cattle, sheep, goats, and pigs. Animals recovered from both diseases seemed to have a durable immunity.—*Onderstepoort J. Vet. Res.* (Oct., 1956): 115.

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**Sweating Sickness.**—This disease, when first recognized in young calves, was believed to be due to a virus but later proved to be caused by a tick-transmitted toxin. The affected animals show dullness, high fever, profuse sweating, with later development of diphtheroid lesions. The etiological agent apparently multiplies in the tick but not in the host and can be transmitted transovarially for five generations. As with tick paralysis, the toxin is apparently produced by the tick while it is engorging, regardless of the infective state of the host.

When the infective ticks (*Hyalomma transiens*) were allowed to feed on calves and pigs, if the feeding period was terminated in less than 72 hours, there was no evidence of a reaction; but if terminated after three to five days, it was followed by an apparent to severe reaction with recovery and some immunity. However, when feeding was not terminated until after six days, there was a severe reaction and 75 per cent fatality. A practical method of control would be to produce immunity by infesting animals with these ticks, then dipping them every four or five days to destroy the feeding ticks. This might eventually eliminate the disease.—W. O. Neitz in *Onderstepoort J. Vet. Res.* (Oct., 1956): 197-203.

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Tuberculosis and histoplasmosis, both of which cause lung cavitation, are frequently found in the same person.—*Am. Rev. Tuberc. and Pulmonary Dis.* (June, 1957).

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**Cortisone Enhances Tuberculosis.**—When the organism which causes bovine tuberculosis was inoculated into the corneas of mice, there was little or no growth on those of mice previously immunized with the same organism. Multiplication occurred in all of the others but it was great-

er, after the first week, in mice treated with cortisone whether or not they had been previously immunized, than in untreated, nonimmunized mice. The cortisone had completely suppressed immunity.—*Brit. J. Pharmacol. and Chemo.* (June, 1957): 240.

## Spinal Tuberculosis in the Dog

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Tuberculosis of the bones, particularly bodies of the vertebrae, is comparatively common in swine and is observed periodically in cattle and horses; but it is apparently rare in dogs and cats, not being mentioned by several authors.<sup>2-4</sup> The following report has several unusual features.

### CASE REPORT

A male German Shepherd, 18 months old, had injured his back in a fight a week prior to the first examination. The dog was lethargic and anoretic but the only abnormality which could be detected was a marked cardiac arrhythmia.

Three weeks later when again seen, he was stiff in the forelegs and had difficulty in climbing stairs. He was still lethargic and not eating well. The cause of the stiffness could not be determined.

Six weeks after the fight, when the dog was again brought to the clinic, he showed slight incoordination in his forelegs, particularly when he first walked after a period of lying down. He was reluctant to mount steps or enter a car.

As he was unable to lower his head in feeding, the bowl had to be held up to his mouth. Forceful raising or lowering of the head produced a marked pain response. On palpating the spinal column, no gross abnormality and no focus of pain could be located. The dog's intelligence appeared to be normal, peripheral sensation was good, and the pedal reflexes were normal.

The pulse, temperature, and respiratory rate were all within the normal range. The dog's appetite remained good but there had

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Dr. B. L. Nestel is with the Department of Agriculture, Jamaica, and Dr. H. M. Nestel is a private practitioner.

Fig. 1—Lateral radiograph showing spinal tuberculosis in the dog. This plate reveals partial destruction of the bodies of thoracic vertebrae 5 to 7, and the loss of marginal definition and of the intervertebral spaces (arrows).



been a steady loss of condition and of coat quality. The urinary constituents were normal and only a low fecal egg count was present. Hematological examinations were not made. No abnormalities were detected on auscultation and palpation of the thoracic and abdominal cavities.

The tentative diagnosis was a spinal lesion in either the lower cervical or the upper thoracic region. Radiographs taken of the spine (fig. 1) showed partial destruction of the bodies of thoracic vertebrae (T.) numbers 5, 6, and 7 with some loss of marginal definition and of the intervertebral disc spaces. There may also have been some involvement of the opposite articular surfaces of T. 4 and T. 8. In the region of T. 5 and T. 6, there was some loss of definition of the costotransverse joints.

Fairly prominent paravertebral shadowing in this region suggested abscess formation, probably due to a chronic infective process, presently active and probably tubercular (Pott's disease).

One cubic centimeter of intradermal tuberculin (0.25 Gm. Koch's old tuberculin) was injected subcutaneously in the neck, and the rectal temperature was taken hourly for 12 hours. Before injection, it was 101.6 F., then it slowly but gradually rose to a peak of 105.1 F. eight hours after injection, unaccompanied by any local or generalized systemic reaction.

The dog was euthanatized for necropsy the day after the tuberculin test. No evi-

dence of a primary lesion was found. The long bones which had not been radiographed were sectioned but none showed evidence of tuberculosis. The only detectable lesion, that involving the thoracic vertebrae, showed a local destruction of bone with the formation of yellow granulation tissue. The affected bones appeared spongy with no detectable cortices and T. 5, 6, and 7 were ankylosed.

Although acid-fast organisms are usually numerous in canine tuberculous lesions, none were found in direct smears.

Material from the lesions was cultured on Jensen's modification of Lowenstein's medium, on which the human strain of *Mycobacterium tuberculosis* grows particularly well. In one month, a good growth was visible. After two months, material from the culture, when injected into rabbits, caused a progressive, generalized, fatal disease, identifying the organisms as of the bovine type. Culture material, injected into guinea pigs, also produced lesions characteristic of tuberculosis and from which acid-fast organisms could be recovered.

#### DISCUSSION

Lesions similar to that described are common in man but seem to be rare in dogs. No primary lesion was found but this is a well recognized phenomenon; it occurred in 13 of one series of 175 dogs with tuberculosis.<sup>1</sup> In this case, it is probable that the organisms spread hematogenously

to the spine from a primary infection in the bowel. Only about 25 per cent of canine tuberculosis is believed to be of bovine origin.<sup>5,6</sup> They arise from the ingestion of contaminated meat or milk.

In Jamaica, tuberculosis in cattle is now rare and all of the milk used in the household from which this dog came was pasteurized. Human tuberculosis is still common here but is rarely due to the bovine strain of the organism. A test on the other 4 animals in the household, and a medical examination of the family and servants, failed to reveal any evidence of tuberculosis. The dog was well fed and kept away from other households. It is possible that the dog was infected before it was imported from England about a year previously. Although that country is rapidly eliminating bovine tuberculosis, it is still much more common there than in Jamaica. It may be that the dog arrived in Jamaica already infected. Against this is the fact that spinal tuberculosis (in man) is usually fairly active and a lesion, such as the one shown here, would not be expected to be more than a year old were it in man.

Under the circumstances, it is difficult to postulate the origin of this infection.

#### SUMMARY

A case of spinal tuberculosis in a dog is described. The lesion, originally thought to be traumatic, was radiologically identified as tuberculosis and confirmed by a tuberculin test and by cultures from the lesion. The organism was typed as the bovine strain; this strain is rare in both animals and man in Jamaica. The origin of the infection was not determined.

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## Some Etiological Findings in Granulomatous Cutaneous Lesions in Dogs

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During a 12-month period, the authors observed a total of 42 dogs with fungus-type, cutaneous lesions. Due to the known prevalence of blastomycosis in this region and the similarity in appearance of these lesions to those of cutaneous blastomycosis,<sup>1</sup> these lesions were critically examined in an attempt to determine the etiological agent or agents.

The lesions were of a suppurative and granulomatous type. Smears of the purulent exudate contained large numbers of eosinophils, macrophages, and lymphocytes, as well as an abundance of phagocytic neutrophils. In the majority of cases, the owners could not state exactly when the lesions had first been evident but, invariably, upon our first examination they were sufficiently extensive to warrant concern.

As in blastomycosis, the primary skin lesion was usually found on an exposed part of the body, particularly the muzzle, head, feet, hocks, legs and, occasionally, the abdomen or back. A purulent exudate was always present and hemorrhage commonly followed the slightest application of pressure. The lesions were definitely circumscribed and raised, and the area within the periphery was usually devoid of hair, hemorrhagic in appearance, and frequently incrusted.

#### METHOD

The following procedure was followed in the examination of these animals:

- The lesions were gently pressed until a purulent discharge appeared.
- Smears of the purulent exudate were made and stained with both Wright's and Gram's stains.
- The purulent material was examined microscopically in wet (10% potassium hydroxide) mounts and, where possible, it was inoculated on the surface of Sabouraud's medium slants and

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<sup>1</sup>Conant, N. F.: Manual of Clinical Mycology. 2nd ed. W. B. Saunders Co., Philadelphia, 1944.

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plates. Hair or scrapings from the lesions were sent to the U. S. Public Health Laboratory at Chamblee, Ga., for examination. The cultures maintained at the clinic were incubated for 21 to 28 days at both room temperature and at 37°C.

4) Blood specimens and serum were sent to the Lexington Clinic laboratory for serological tests for blastomycosis.

The microscopic, cultural, and serological findings are shown (table 1). Where the tests were not complete, the animal owner had either refused to cooperate or the dog was not returned for laboratory work.

#### DISCUSSION

As indicated in table 1, the majority of the affected dogs were presented for examination during the months of October, November, and December. In 12 of 14 animals where *Trichophyton mentagrophytes* was discovered, by microscopic examination of the purulent exudate or by culture or both, the serological test for blastomy-

crosis was positive. The titers ranged from 1:4 to 1:512.

This raises a question whether there is any relationship between certain mycological agents, particularly *T. mentagrophytes* and blastomycosis, or whether the association lies purely in the serological reaction. There was also a complement-fixation titer of 1:4 and 1:16 in 2 of 3 animals in which *Microsporum canis* was found. *Microsporum gypseum* and *M. canis* were the only mycological agents identified other than *T. mentagrophytes*. A conclusion concerning blastomycosis can not be drawn as to the significance of the positive serological reactions in these animals with these infections. However, one might speculate that there are many other instances involving different mycological agents where positive serological reactions for blastomycosis occur.

TABLE I—Cultural, Microscopic, and Serological Data from Dogs Having Cutaneous, Blastomycotic-like Lesions

Date	Breed	Site of lesion	Microscopic examination Stained smear, Wright's, and Gram's procedures	Culture Sabouraud's medium	Complement- fixation for blastomycosis
Nov., 1955	Mixed	Muzzle	Negative	Not done	Positive 1:64
Nov., 1955	Weimaraner	Muzzle, foot	B. dermatitidis*	B. dermatitidis	Positive 1:128
Jan., 1956	Cocker Spaniel	Muzzle	Negative	Not done	Positive 1:64
Jan., 1956	Collie	Muzzle	B. dermatitidis*	Not done	Positive 1:64
Jan., 1956	Setter	Muzzle, head	Negative	Negative	Negative
Feb., 1956	Poodle	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:64
Feb., 1956	Poodle	Head	Negative	Negative	Negative
March, 1956	Beagle	Muzzle	Negative	Negative	Negative
April, 1956	Dachshund	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:256
April, 1956	Pointer	Muzzle	Negative	Negative	Negative
June, 1956	Boxer	Muzzle	Negative	Negative	Negative
June, 1956	Spaniel	Muzzle	Negative	Negative	Negative
July, 1956	Collie	Head	Negative	Negative	Negative
July, 1956	Hound	Muzzle	T. mentagrophytes	T. Mentagrophytes	Negative
Sept., 1956	Boxer	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:512
Sept., 1956	Hound	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:64
Oct., 1956	Weimaraner	Muzzle, foot	T. mentagrophytes	T. Mentagrophytes	Negative
Oct., 1956	Boxer	Back	T. mentagrophytes	T. Mentagrophytes	Positive 1:64
Oct., 1956	Terrier	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:64
Oct., 1956	Cocker Spaniel	Muzzle, head	M. canis	M. canis	Positive 1:16
Oct., 1956	Collie	Muzzle, head	Negative	Negative	Positive 1:2,048
Oct., 1956	Boxer	Muzzle	Negative	Negative	Positive 1:4
Oct., 1956	Dachshund	Muzzle	M. canis	M. canis	Positive 1:4
Oct., 1956	Dachshund	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:4
Oct., 1956	Dachshund pup	Leg, belly	T. mentagrophytes	T. Mentagrophytes	Not done
Oct., 1956	Dachshund pup	Leg, belly	T. mentagrophytes	T. Mentagrophytes	Not done
Oct., 1956	Boxer	Muzzle	Negative	Negative	Not done
Oct., 1956	Cocker Spaniel	Leg	Negative	Negative	Not done
Nov., 1956	German Shepherd	Muzzle, leg	T. mentagrophytes	T. Mentagrophytes	Positive 1:4
Nov., 1956	Collie	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:64
Nov., 1956	Boxer	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:4
Nov., 1956	Cocker Spaniel	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:4
Nov., 1956	Basset Hound	Muzzle	Negative	Negative	Negative
Nov., 1956	Dachshund	Muzzle	Negative	Negative	Not done
Nov., 1956	Boxer	Muzzle	Negative	T. Mentagrophytes	Not done
Nov., 1956	Cocker Spaniel	Muzzle	T. mentagrophytes	T. Mentagrophytes	Not done
Dec., 1956	Cocker Spaniel	Muzzle	T. mentagrophytes	T. Mentagrophytes	Positive 1:16
Dec., 1956	Mixed	Muzzle	Negative	M. canis	Negative
Dec., 1956	Mixed	Muzzle, back	M. gypseum	M. gypseum	Not done
Dec., 1956	Mixed	Head, foot	T. mentagrophytes	T. Mentagrophytes	Negative
Dec., 1956	Boxer	Muzzle	Negative	T. Mentagrophytes	Not done
Dec., 1956	Mixed	Back	Negative	T. Mentagrophytes	Not done

\*Blastomycetes seen in 10 per cent potassium hydroxide mounts from only 2 of the 42 dogs.

It would seem that mycological infections of this particular type were more prevalent during the late fall months; also, that *T. mentagrophytes* is responsible for most of the fungal infections of dogs in this area.

#### SUMMARY

Of the smears taken from the circumscribed, suppurative, and granulomatous lesions, primarily on the muzzle, head, feet, and legs of 42 dogs, 23 showed infections as follows: *Trichophyton mentagrophytes*, 18; *Blastomyces dermatitidis*, 2; *Microsporum canis*, 2; and *M. gypseum*, 1.

Of the cultures made from 39 dogs, 26 showed growth as follows: *T. mentagrophytes*, 21; *B. dermatitidis*, 1; *M. canis*, 3; and *M. gypseum*, 1.

Complement-fixation tests for blastomycosis on 32 dogs were positive in 20, although 18 of these showed no microscopic or cultural evidence of *B. dermatitidis*. Of these 18 dogs, *T. mentagrophytes* was recovered from the lesions of 12 with titers ranging from 1:4 to 1:512; *M. canis* was recovered from 2; and no organisms were found in 4.

Although not necessarily responsible for the lesions, *T. mentagrophytes* was found in 22 of the 42 dogs.

**Effect of Hog Cholera Virus on Endocrine Glands.**—Microscopic examination of the thyroid, thymus, pituitary, pineal, pancreas, testicles, and ovaries from 102 pigs, 2 to 4 months old, infected with hog cholera virus, showed pathological changes similar to those in the lymphoid tissue, digestive tract, and nervous system. These changes have little significance and little value in diagnosis. If a young pig should survive, damage to these glands might affect its growth and physical condition.—*Archiv. Exper. Vet.-med.*, 10, (1956): 189 (abstr. in German Sci. Bull. (June, 1957): 3).

**Changes in the Nervous System in Hog Cholera.**—A study of the nervous system of swine with hog cholera indicates that changes occur in the ganglia, which are similar to those found in swine paralysis. Similar but less marked changes occur in the ganglia of healthy swine. They are nonspecific and are considered to be secondary changes. To what extent they in-

fluence the clinical picture in disease needs further investigation.—*Monatsh. f. Vet.-med.* (March 1, 1956): 108.

**Induced Toxoplasmosis in Dogs.**—A *Toxoplasma gondii* strain, isolated from a dog, was used to inoculate 30 dogs experimentally and infection was achieved as follows: in 2 of 6 dogs inoculated by mouth, 4 of 6 inoculated subcutaneously, 3 of 5 inoculated intraperitoneally, in 10 of 10 inoculated intracranially, and in none of 3 inoculated intravenously. Three of the 19 infected dogs developed acute toxoplasmosis.—*Arch. Inst. Pasteur d'Algérie*, 35, (1957): 29.

**Stages of Toxoplasmosis Infection.**—When infective material from rats with toxoplasmosis was injected into mice, the infection developed in three stages: the acute phase with parasitemia, lasting up to three weeks; the subacute stage, the organism being found chiefly in the liver and brain; and, about two months later, the chronic stage, when parasites were found only in the brain. Titers to the dye test were highest in the second and third weeks after infection, then gradually declined.—*Vet. Bull. (May, 1957)*: Item 1420.

**Treatment of Toxoplasmosis in Rabbits and Mice.**—In treating experimental toxoplasmosis in rabbits, sulfadimidine alone is of little value since it is rapidly acetylated by most rabbits. Sulfathiazole is acetylated less rapidly and is effective in all rabbits.

In mice, sulfadimidine was effective but relapses were common. Pyrimethamine was effective and prevented the carrier state but this required near toxic levels. The combination of sulfadimidine and pyrimethamine, which were synergic, in doses well below the toxic level controlled the acute infection and prevented relapses and the carrier state.—*Bri. J. Pharmacol. and Chemq.* (June, 1957): 185 and 189.

**Caseous Lymphnoditis in Sheep.**—An enzootic disease of sheep, in Poland, characterized by caseous lymphnoditis, mostly in lambs, yielded *Pasteurella multocida* in the pus. Penicillin therapy failed, but recovery followed the use of streptomycin (0.5 Gm.) twice daily for two or three days.—*Med. Weteryn.* (Feb. 1957): 75.

## Liver Fluke (*Metorchis conjunctus*) in a Dog from South Carolina

HELEN E. JORDAN, D.V.M., M.S., and  
WILLIAM T. ASHBY, D.V.M.

Athens, Georgia

Infections with the liver fluke (*Metorchis conjunctus*, Cobbald, 1860) have been reported in dogs, cats, foxes, mink, and raccoons in Canada, Newfoundland, and Maine.<sup>2-4</sup> The presence of the opisthorchid trematode in a dog in the Southeast is considered noteworthy.

In view of the rarity of this parasite (fig. 1) in domestic animals within the United States, a brief description with notes on its life cycle may be in order.

The body is linguiform and measures from 1.0 to 6.6 mm. in length and 0.59 to 2.5 mm. in width. The sizes of the specimens are roughly proportional to the size of the host, with the largest specimens from dogs and raccoons and the smallest from the mink.<sup>1</sup> The suckers are of about the same size (0.24 to 0.09 mm. in diameter), with the ventral sucker anterior to the beginning of the second third of the body length. The intestinal cecum almost reaches the posterior end of the body. The two testes are spherical in shape, diagonally arranged, and situated in the anterior portion of the terminal third of the body length. The ovary lies just anterior to the testes. The yolk glands are lateral to the cecum, most in the anterior half of the body, and the uterus fills most of the space between the cecum, from the lower ovary to just anterior to the ventral sucker. The distinctly operculated, yellowish brown eggs are 22 to 32  $\mu$  long and 11 to 18  $\mu$  wide.

The life history of *M. conjunctus* involves a snail (*Amnicola limosa porosa*) as the first intermediate host and the common sucker fish (*Catostomus commersonii*) as the second intermediate host. Infection in the definitive host occurs when a susceptible host ingests the infected, second intermediate host.

**Case Report.**—A female hound, 4 years old, had gradually become debilitated and, on Jan. 8, 1956, showed ascites and an increased heart rate, but a weak pulse. Although therapy with digitalis and a sodium-free diet temporarily alleviated this condition, the dog's general condition continued to decline and, after six months, the owner requested euthanasia.

At necropsy, the liver was enlarged and turgid, with the gallbladder distended and



Fig. 1—Sketch of adult *Metorchis conjunctus* (liver fluke) recovered from dog's liver (after fixation).

the bile ducts (fig. 2) containing more than 100 of these flukes. Microscopic examination revealed marked cirrhosis, proliferation of the bile ducts, acute nephritis with swelling, and a degeneration of the myofibrillae of the cardiac musculature.

With regard to the source of infection, the dog reportedly had not been out of the state; however, it probably ate raw fish from local streams. Since the common sucker is the infective intermediate host for this fluke, the dog probably became infected from ingestion of this fish.

### References

- Cameron, T. W. M.: The Morphology, Taxonomy, and Life History of *Metorchis conjunctus*



Fig. 2—Bile duct with cross section of *Metorchis conjunctus* (liver fluke) in situ. H & E stain; x 100.

From the School of Veterinary Medicine, University of Georgia, Athens. Dr. Ashby is a general practitioner in Barnwell, S. Car.

(Cobbold, 1860). Canad. J. Res., 22, (1944): 6-16.

<sup>3</sup>Cameron, T. W. M., Parnell, I. W., and Lyster, L. L.: The Helminth Parasites of Sledge-Dogs in Northern Canada and Newfoundland. Canad. J. Res., 18, (1940): 325-332.

<sup>4</sup>Grice, H. C., and Hutchison, J. A.: Obstructive Jaundice Ascribed to *Metorchis Conjunctus* in a Cat with a Bifid Gallbladder. J.A.V.M.A., 130, (1957): 130-132.

<sup>5</sup>Meyer, M. C.: The Presence of *Metorchis conjunctus* in Maine. J. Parasitol. Sect. 2, 35, (1949): 39.

## Palsy Syndrome in Tiger Cubs

L. E. FISHER, D.V.M.

Berwyn, Illinois

Three tiger cubs, 2 males and 1 female, weighing about 2 lb. each, were born in Lincoln Park Zoo, Chicago, on July 16, 1956. They were immediately removed from their mother as she seemed determined to kill them.



Fig. 1—Tiger cubs when 6 weeks old, shown with their incubator.

The cubs were placed in a baby incubator and fed with nursing bottles every three hours. The formula, consisting of milk, water, Borden's Esbilac, and vitamin supplements, was varied when indicated to counter the frequent occurrence of diarrhea.

Dr. Fisher is a small animal practitioner in Berwyn, Ill. The author thanks Drs. S. Schulman, University of Chicago Medical School; Albert Scheitzer, Raymond Clasen, Bruce Taylor, and David Brown of the Presbyterian Hospital in Chicago, Ill.

Their eyes opened when they were 14 days old and, until they were 3 weeks old, except for the soft stools, the cubs seemed normal and gained weight steadily but slowly.

As the cubs began to move more independently, a palsy was noticed, but this was not considered abnormal until they were a month old when the debility was marked in the female and in 1 male. These 2 cubs showed a rapid bobbing of the head as they walked and 1 showed nystagmus. The other male did not seem to be as greatly affected. The generalized tremors did not persist during sleep. When they were a month old, it was also noticed that the corneas of their eyes were not clear and in all 3 there was some mineral-like deposit in the posterior portion of the lens.



Fig. 2—Two of the young tigers when about 6 months old and fully recovered.

Rectal temperatures ranged from 100 to 102 F.

The tentative clinical diagnoses, which were suggested by consultants, ranged from vitamin deficiencies to toxoplasmic infection. ACTH and vitamin B<sub>12</sub> injections were given daily for five days, and vitamin E injections were initiated. No laboratory tests were made because improvement was soon noticeable and, in sev-

eral weeks, the cubs seemed to be approaching normal behavior (fig. 1). Their diet was gradually broadened to include fresh meat, mechanically and thoroughly macerated, along with beef viscera, blood, and more vitamin and mineral supplements.

Feline distemper serum was administered at 18 days of age and twice repeated at ten-day intervals. After that, small repeated doses of feline distemper vaccine were given at weekly intervals for four weeks.

When 5 months old, the average weight of the 3 cubs was approximately 50 lb., and at 7 months, it was approximately 65 lb. (fig. 2). At 10 months, the young tigers seemed entirely normal and weighed about 100 lb. each.

**Sedation and Body Temperature.**—Chlorpromazine and reserpine reduce locomotor activity and prolong pentobarbital hypnosis in mice in proportion to the fall in body temperature which they produce. Since neither drug reduces activity significantly unless the temperature is reduced, it is possible that they act by virtue of ability to lower body temperature. The prolonging of hypnosis may be due to a reduced rate of detoxication resulting from this lowered temperature. Neither drug produces apparent sedation of mice at a room temperature of 32 C. (89.6 F.).

However, at a room temperature of 32 or 36 C. (96.7 F.), chlorpromazine potentiates pentobarbital hypnosis while reserpine does not. Therefore, the action of chlorpromazine is not related to a hypodermic effect.—*Brit. J. Pharm. and Chemo.* (June, 1957) : 245.

**Steroids and Involution of the Thymus.**—The activity of corticosteroids in causing involution of the thymus of young rats provides a quick and simple screening test for their biological activity. Of the steroids investigated, those possessing marked thyromolytic activity were also those with therapeutic properties.—*Brit. J. Pharmacol. and Chemo.* (June, 1956) : 133.

**Magnetometer Records Gastric Motility.**—Since tubes or wires issuing from the mouth or nose constitute a persistent noxious stimulus, in studying human gastric motility, a magnetometer has been devised.

The subject swallows a magnet about the size of a vitamin capsule, then reclines on a cot while a detector, 18 inches below the subject's stomach, senses and records stomach activity from motions of the magnet. The device is also used to detect patterns of movements in small laboratory animals with magnets fixed under their skin.—*Science* (May 17, 1957) : 990.

**Anthrax in Sheep.**—Anthrax, with a mortality of 46 per cent, occurred in a flock of sheep in Germany. Most of the animals showed apoplectic rather than characteristic clinical signs. The chief lesions were a large hemorrhagic spleen and a serous hemorrhagic edema of the subcutis. The source of infection was probably a grass plot in which anthrax cadavers had been buried decades before.—A. Konig in *Deutsche tierärztl. Wchnschr.* (Dec. 1, 1956) : 474.

A fowl cholera vaccine was improved by the addition of aluminum hydroxide-gel to the Pasteurella bouillon cultures. It was further improved by reducing the formalin content and increasing the germ concentration.—*An. Inst. Seruri Si Vaccin. Pasteur Bucuresti*, 1, (1956) : 220.

**Spray Prevents Cannibalism and Keeps Out Unwelcome Birds.**—In an experiment, at Cornell University, to determine whether chickens have taste sense, 200 flavored chemicals were tested. Nine were so repugnant that they stopped cannibalism for two weeks to two months. Other possible uses of the repellent are: spraying public buildings to keep away starlings and pigeons; spraying ripening berries and fruits (not yet approved by the F.D.A.); and spraying ripening grains or seed grains to prevent loss to birds.—*Farm J.* (July, 1957) : 31, 60.

In every flock of chickens, 1 bird can peck all others with impunity and the next in order can peck all others except the first. With birds, the highly colored males are dominant but when the most timid females are colored like males, they then can dominate many other females. With cattle, seniority seems to determine the order of dominance.—*Sci. News Letter* (July 13, 1957) : 32.

# Nutrition

## Field Services of a Feed Company Veterinarian\*

The challenge in this type of work is wider in scope than most people realize. It requires a good knowledge of veterinary medicine, management, breeding, and nutrition as well as of salesmanship (not actually making sales). One has to keep the salesman of the territory, the feed company, the farmer, and the local veterinarian in mind at all times.

Most of the problems of service deal with management of the herd or flock. One of the biggest problems is internal parasites. The American public wants everything by the "push button" method which means herd or flock treatment. This is better than no treatment but is not equal to individual treatment of heavily parasitized animals. To make the best of this situation, the animals or birds should be grouped according to size and dosed adequately. The procedure should be repeated in 30 to 40 days if necessary.

The salesmen for most companies are trained to sell minerals and supplements and to recognize some of the minor problems. They are trained not to perform necropsies or to make diagnoses. They are never permitted to treat or prescribe for acutely diseased animals or poultry. This improves relationships in the field for all concerned.

\*Prepared by Dr. M. I. Thiele, Marshalltown, Iowa, in cooperation with the American Association of Veterinary Nutritionists.

**Antibiotics in Food.**—Over 440 tons of penicillin was produced in 1956 and was marketed in 150 preparations. Perhaps 10 per cent of people develop an allergy to penicillin; many more than to most antibiotics. There are no known cases of reaction to penicillin in milk; however, in a recent survey, penicillin was found in concentrations up to 0.55 units per milliliter in 5.9 per cent of 1,706 milk samples from all states. Dyes may eventually be added to color penicillin containing milk. The other antibiotics used in mastitis remedies do not create a public health problem.

Antibiotic residues are not found in animals to which they are fed as growth stimulants. Nor are they found, after cooking, in dressed poultry immersed for two hours in iced water containing 10 p.p.m. of the tetracyclines. Antibiotics create a

much less serious food-processing problem than do equipment lubricants and cleaners, which may contaminate food.—*Am. J. Pub. Health* (June, 1957): 701.

**To Tag Penicillin in Milk with a Dye.**—A dye which will color milk up to 72 hours, and which is nontoxic to a cow's udder, is being sought by the Food and Drug Administration for addition to mastitis remedies which contain penicillin. An early sulfonamide that colors milk red is satisfactory but only for 30 hours. After July, 1957, the penicillin content of a packet will be limited to 100,000 units but other antibiotics can be added.—*Sci. News Letter* (June 29, 1957): 403.

**Sanitation and Antibiotic-Fed Calves.**—When chlortetracycline was either fed or injected intramuscularly in calves 12 weeks old, weight gains and feeding efficiency were increased whether they were raised in new and uncontaminated or in old quarters. Growth response was detected earlier in those treated orally than in those receiving injections; and earlier in the new barn (2 weeks) than in the old (4 weeks). There was no significant difference in leukocyte counts in the treated and control calves either in old or new quarters.—*J. Dai. Sci.* (Jan., 1957) : 50-55.

**Fresh Cud vs. Rumen Culture Preparations.**—The effect of early rumen flora development in calves was investigated. Cud-inoculated calves developed a good cellulolytic flora in two weeks whereas noninoculated calves and those given dried preparations showed a retardation in flora development. There were no beneficial effects of the dried preparation in preventing calf scours.—*G. W. Wieringa in Overdruk Uit, Tijdschr. voor Diergeesk.* (March 15, 1956): 242.

When barley stored for three years was compared with fresh barley, it had an equal feed efficiency value and produced equal gains when fed to swine with a proper supplement (equal parts casein, soybean oil meal, and cottonseed meal). Storage did not affect the nutritive value of the protein for rats when, in four successive years, barley was fed both "before and after" storage.—*J. Nutr.* (March 10, 1957): 43.

## Changes in General Veterinary Practice

GUEST EDITORIAL (Requested)

There have been many changes in the practice of veterinary medicine during the past 40 years. If, immediately following his graduation in 1917, a veterinarian had imbibed a double dose of Rip Van Winkle's potion and awakened 40 years later, he would find himself inadequately prepared to practice today.

He would be confused by such terms as leptospirosis, listerellosis, parakeratosis, hyperkeratosis, dystrophic rhinitis, ketosis, and eperythrozoönosis. He would have even more trouble spelling and pronouncing them than we do. These, with the many antibiotics, sulfonamides, and corticosteroids, as well as radioactive isotopes and new biological products, would probably drive him at once to modern tranquilizers.

However, the activities of most other professions and trades have changed as much as ours. If a mechanic had been presented 40 years ago with a 1957 model car, would he even have been able to find the spark plugs? The 1917 veterinarian would be in a similar predicament. Although his models (animals) have changed slightly, if at all, many disease problems would be new.

The veterinary profession has adapted itself to these changes. It has met the challenge of new diseases and has improved the means of diagnosis and of therapy for the old familiar diseases. Credit for these improvements must be given to the research workers who sought new information; to the drug and biological industry men who developed the products needed; and to the practitioners whose observations and requests for enlightenment often stimulated research, and who evaluated new theories and therapeutic agents in the field.

The veterinary colleges have changed their curriculums and teaching methods to keep pace with advancing veterinary science. Many have also conducted conferences and short courses to keep practitioners informed. Extension veterinarians in many states have taken this information to the practitioner in the field.

Veterinary medical associations, local, state, and national, have held meetings where outstanding practitioners, educators, and scientists have unselfishly given their time to share their findings with others.

Veterinary journals publish original articles and reviews of literature pertain-

ing to all phases of veterinary medicine. Also, a wealth of material is received from commercial firms, giving information on new drugs and their uses.

### MODERN GENERAL PRACTICE

Let us see how these changes have affected veterinary practice.

*On the credit side* are the following:

Improved roads and automobiles have made it possible to make many more calls in greater comparative comfort.

Modern offices and hospitals have replaced the livery barn and dingy back rooms which veterinarians once used.

Stainless steel and chrome-plated instruments in well-arranged cases have outmoded the rusty, tarnished steel equipment.

Drugs are now vastly more efficient, better packaged, and easier to administer.

Ill-smelling antiseptics have been replaced by other effective ones which don't herald our presence a city block away.

Portable chutes and equipment make animal restraint faster, easier, and safer.

We now sit in air-conditioned rooms to view clinical demonstrations on closed-circuit television instead of standing in a crowd to get a rare glimpse of procedures.

Additional income is derived from disease control and regulatory work.

*On the debit side* are the following:

In many areas there has been a reduction in the numbers of clients and of livestock. Many small farms, where grain was marketed as livestock products, have been consolidated into large grain farms with little or no livestock. This change has been stimulated by power machinery, the high cost of farm labor, and government subsidies on grain.

The practitioner's income is becoming increasingly dependent upon government fees; the farmer will probably have more government-provided veterinary service as a subsidy to compensate him for the price squeeze forced upon him.

Many regret the resulting loss of independence; in regulatory work we must follow instructions and regulations, make reports, and accept the fees provided.

Yes, this is a changing world. The Dodo bird couldn't make it, but we feel sure that the veterinarian will.—*Frank B. Young, D.V.M., Waukee, Iowa.*

# Current Literature

## ABSTRACTS

### A Modified Assay for Vitamin B<sub>12</sub>

The *Euglena gracilis* var. *bacillaris* assay of Ross gave a low percentage recovery of vitamin B<sub>12</sub> added to swine serum. The assay was modified by adding potassium cyanide to the serum, increasing the heat treatment, and centrifuging the resulting mixture to remove the coagulated protein and other precipitates. Growth of the assay organism was measured by titrating the alkali production with a Beckman model K automatic titrator. The recovery of vitamin B<sub>12</sub> added to swine serum was 100 per cent and the determination of total serum vitamin B<sub>12</sub> was improved.—[G. L. Frederick: *A Modified Assay for Vitamin B<sub>12</sub> in Swine Blood Serum.* Am. J. Vet. Res., 18, (July 1957): 481-483.]

### Pasteurella Multocida Immunization of Chickens and Mice

A solution of the capsular polysaccharide, which was removed from the encapsulated virulent dissociant of *Pasteurella multocida* by heat treatment at 57 C. for one hour, immunized chickens and mice against challenge with the homologous strain.

A living or formalin-killed vaccine of the unencapsulated avirulent dissociant was protective only for chickens. A killed vaccine of a heterologous type protected neither host. Possible explanations for this host difference in the immune response and its relationship to antigenic differences of the dissociants are discussed.—[Katherine E. Yaw and J. C. Kakavas: *A Comparison of the Protection-Inducing Factors in Chickens and Mice of a Type 1 Strain of Pasteurella Multocida.* Am. J. Vet. Res., 18, (July, 1957): 661-664.]

### Penicillin V in Cattle

In normal, adult, milking cows, single intramuscular doses of 3 million units of benzathine penicillin V in aqueous suspension produced significant penicillemia for 96 hours and significant and uniform penicillactia for 48 hours. Benzathine penicillin G produced penicillemia for 144 hours with negligible penicillactia; procaine penicillin G produced significant penicillemia and penicillactia for 24 hours.

The results suggest penicillactia may result from the active participation of mammary tissue rather than mere blood circulatory spill.—[C. J. Hollister, R. A. Huebner, W. B. Boucher, and T. DeMott: *Parenteral Benzathine Penicillin V in Cattle.* Am. J. Vet. Res., 18, (July, 1957): 584-586.]

### Piperazine for Ascarid-Infected Pigs

Fifteen ascarid-free pigs were separated into three groups of 5. One group was left on normal feed while a second group (principals) was given 1/5 of a therapeutic dose of polymeric piperazine-l-carbodithioic acid per day (320 mg./16-kg. pig)

in the feed one week prior to natural ascarid infection. The third group was raised on normal feed as ascarid-free controls in a tile and concrete room. Ascarid-infected pigs and controls were killed at three, four, five, and six weeks for comparative purposes. The principals were continued on the drug feed mixture throughout the experiment. This level of drug did not prevent migration of worm larvae nor was it capable of completely removing larvae from the 5 principal pigs. One can not predict from these data what effect this continuous level would have on mature worms.—[W. D. Lindquist: *The Use of Low Level Piperazine on Pigs Naturally Infected with Ascaris Lumbricoides.* Am. J. Vet. Res., 18, (July, 1957): 508-510.]

## BOOKS AND REPORTS

### Medical Manual of Chemical Warfare—A Symposium

Recent developments in chemical warfare, and newer concepts in therapeutics and experimental physiology, have necessitated the publication of this revised, third American edition of the symposium.

In this edition, detailed descriptions of dangerous gases and the handling, recognition, and early treatment of gas casualties are given. Particular attention is devoted to war gases, especially the newer nerve gases.

The book contains illustrations, an index, and tables, including a table dealing with the effects of war gases on food and water.—[*Medical Manual of Chemical Warfare—A Symposium.* Edited by a board of editors, chairman, Major-General A. Sachs. 86 pages. Chemical Publishing Co., Inc., 215 Fifth Ave. New York, N.Y. 1956. Price \$4.00.]

### Comparative Anatomy of the Eye

This book is, as the author states, a review of comparative anatomy of the eye designed for students, research graduates, and teachers. It is an excellent treatise on the subjects of the comparative and developmental anatomy, with a good deal of the basic physiology explained. Anatomy and function of the eye types found in the various groups of vertebrates are discussed and compared. As it was written with the academic approach, it is, from a clinician's standpoint, lacking in much of the practical information which would be of most value in diagnosis and treatment of clinical eye disease. For those individuals, however, who have more than a passing interest in eye diseases, this book contains a great deal of information which enables the reader to obtain a much better understanding of ocular function and pathology. It should be a valuable addition to the library of one with an interest in ophthalmology.—[*Comparative Anatomy of the Eye.* By Dr. J. H. Prince. 379 pages. Charles C. Thomas, Springfield, Ill. 1956. Price \$8.50.]—HUGH D. SIMPSON.

# THE NEWS

## Dr. Hutchings to Head Purdue's New School of Veterinary Medicine

A School of Veterinary Science and Medicine has been approved by the Purdue University board of trustees following authorization by the Indiana legislature. Dr. L. M. Hutchings, head of the Purdue Department of Veterinary Science, has been named dean of the new school, which will be the eighteenth of its kind in the United States.

The school will be equipped to admit not more than 50 students in each beginning class. If facilities and finances permit, a small class will be enrolled in the fall of 1958; otherwise, classes will not start until the autumn of 1959.

New buildings for the veterinary school will be constructed at the south edge of the campus. Hospital and clinical facilities with an ambulatory clinic will be provided for large and small animals.

The curriculum will consist of a six-year program divided into two parts. A two-year pre-veterinary program comparable to premedical requirements, with some agricultural courses added,

strong graduate student enrollment. Research, extension, and public services of the department will continue under the director of agriculture.

The new school will round out a program started at Purdue in 1887. At that time, the Department of Veterinary Science was established to "prevent the enormous losses from animal disease." It was one of the first agricultural departments to be organized at the university.

Dr. Hutchings joined the staff at Purdue in 1942; he was promoted to professor in 1947 and became head of the Department of Veterinary Science in 1950. A graduate of the University of Maine (B.S., 1937), Dr. Hutchings received his D.V.M. from Michigan State University in 1940 and the M.S. degree in 1942. In 1947, he was granted a Ph.D. degree in pathology from Purdue University. His special field of research is brucellosis.

In 1947, Dr. Hutchings won the Sigma Xi Research Award. He is a member of the sections on animal health, brucellosis, and leptospirosis in man and animals of the National Research Council, as well as the Committee for Eradication of Hog Cholera of the U.S. Livestock Sanitary Association. Dr. Hutchings also serves on the World Health Expert Panel on brucellosis and is the member of the AVMA Executive Board from District III (Indiana, Illinois, and Wisconsin.)



Dr. L. M. Hutchings

will continue to be taught in the School of Agriculture. The second part of the program is a professional school curriculum of four years and is the newly authorized area of study. All basic requirements of the AVMA will be met for professional accreditation. The new school will give the D.V.M. degree.

Research programs in animal diseases and animal pathology will be continued to attract a

## Dr. Ralph L. Kitchell Awarded a Special Research Fellowship

Dr. Ralph L. Kitchell, head of the Division of Veterinary Anatomy at the School of Veterinary Medicine, University of Minnesota, has been granted a special research fellowship from the National Institute of Neurological Diseases and Blindness, U. S. Public Health Service, for a year of research and study, beginning August 30, 1957.

He will spend the first nine months working under Professor Y. Zotterman, Department of Physiology, Royal Veterinary College, Stockholm, Sweden. The next three months, Dr. Kitchell will study with Professor Amoroso, Department of Physiology, Royal Veterinary College, London.

The title of the research project that Dr. Kitchell will be concerned with is the "Electrophysiological Study of Vagal Afferents from the Reticulum and Rumen and Their Central Projections in the Goat."

## National Science Foundation Announces Availability of Publications

The National Science Foundation publications are available to all scientists who have need for them. Lists of publications are available from the foundation. Requests should be addressed to the Publications Office, National Science Foundation, Washington 25, D. C.

**Veterinarians Awarded Advanced Degrees**

During the 1956-1957 academic year, the following advanced degrees were granted to veterinarians engaged in postdoctoral studies:

Name of student	Title of thesis	Major professor	Dept. and school	Date granted
AWARDED MASTER OF SCIENCE (M.S.) DEGREES				
Frank G. Fielder, B.V.Sc.	Dermalon Skin Sutures—A Comparison	A. G. Danks	N.Y. State Vet. College, Cornell Univ.	June, 1957
T. F. Zweigart, Jr., D.V.M.	Study of Some Physiological Properties of Micrococci as Related to Pathogenicity for the Bovine Udder	A. L. Kleckner	Bacteriology Dept., College of Arts & Sciences, Univ. of Georgia	June, 1957
Aaron H. Groth, Jr., D.V.M.	Suppressed Endothelial Cell Differentiation in the Ribs of Immature Iowa Swine	E. A. Benbrook	Vet. Pathol. Div. of Vet. Med., Iowa State College	July, 1957
Charles Hatch, M.R.C.V.S.	Effect of Fleece Contaminants on Activity of Nine Blowfly Larvicides	E. A. Benbrook	Vet. Pathol. Div. of Vet. Med., Iowa State College	June, 1957
Vaughn A. Seaton, D.V.M.	Histopathology of Pulmonary Adenomatosis in Iowa Cattle	William S. Monlux	Vet. Pathol. Div. of Vet. Med., Iowa State College	June, 1957
Harry D. Anthony, D.V.M.	Bacteriological and Pathological Studies of Bovine Keratitis	M. J. Twiehaus	Vet. Pathol. School of Vet. Med., Kansas State College	May, 1957
Om Parkash Malhotra, B.V.Sc.	Study of Incidence of the Neoplasms with Particular Reference to the Female Genital Tract of Animals	F. H. Oberst	Surgery & Med. School of Vet. Med., Kansas State College	May, 1957
James R. Reif, B.S. D.V.M.	Distribution of Cathomycin in the Bovine when Administered Parenterally	G. H. Conner	Surgery & Med., College of Vet. Med., Mich. State Univ.	(1957 summer session)
Jose Britto Figueiredo, D.V.M.	Comparison of the California Mastitis Test with the Other Commonly Employed Diagnostic Tests	L. C. Ferguson	Microbiology & Pub. Health Dept. College of Vet. Med., Mich. State Univ.	(1957 summer session)
D. E. Rodabaugh, D.V.M.	Histopathology in the Gastro-intestinal Tract of Sheep Infected with Nematodes Surviving Phenothiazine—Salt Prophylaxis	Cecil Elder	Vet. Pathol. School of Vet. Med., Univ. of Missouri	June, 1957
Howard S. Garner, D.V.M.	Compilation of Literature of Helminth Parasites of Monkeys and Apes	Fleetwood R. Koutz	Vet. Parasitology College of Vet. Med., Ohio State Univ.	June, 1957
Adalbert Koestner, D.V.M.	Veterinary Neuropathology	Clarence R. Cole	Vet. Pathol. College of Vet. Med., Ohio State Univ.	June, 1957
Thomas E. Murchison, D.V.M.	Histochemical Studies in Vet. Pathology	Clarence R. Cole	Vet. Pathol. College of Vet. Med., Ohio State Univ.	June, 1957
Svend W. Nielsen, D.V.M.	Canine Mastocytoma	Clarence R. Cole	Vet. Pathol. College of Vet. Med., Ohio State Univ.	June, 1957
George A. Elliot, D.V.M.	Focal Necrosis in the Cerebral Cortex of the Bovine: A Histopathologic Study	Evan L. Stubbs	Grad. School of Arts and Sciences School of Vet. Med., Univ. of Pa.	June, 1957
Theodore Yerasimides, D.V.M.	Comparative Pathology of Inflammation.	Herbert Ratcliffe	Grad. School of Arts and Sciences College of Vet. Med., Univ. of Pa.	June, 1957
Leon W. Gibbs, D.V.M.	Investigation of a Necropsy Procedure of the Parathyroid Glands of the Ox by a Study of the Anatomical Relationships	H. A. Smith	Vet. Pathol. School of Vet. Med., Texas A. & M.	May, 1957

Name of student	Title of thesis	Major professor	Dept. and school	Date granted
<b>AWARDED MASTER OF SCIENCE (M.S.) DEGREES—Continued</b>				
Leland C. Grumbles, D.V.M.	Etiology of Infectious Sinusitis of Turkeys	J. P. Delaplane	Vet. Microbiology School of Vet. Med., Texas A. & M.	May, 1957
James E. Neal, D.V.M.	Composition of the Indirect Complement Fixation Test, the Direct Complement Fixation Test, and Rapid Plate Agglutination Test for the Ornithosis Antibody in Turkey Sera	J. P. Delaplane	Vet. Microbiology School of Vet. Med., Texas A. & M.	May, 1957
<b>AWARDED DOCTOR OF PHILOSOPHY (Ph.D.) DEGREE</b>				
Charles M. Barnes, D.V.M.	Quantitative Study of the Reticuloendothelial System in the Developing Chicken Embryo	Logan M. Julian	Comp. Pathol. School of Vet. Med., Univ. of Calif.	June, 1956
Walter S. Tyler, D.V.M.	Nature of Short-Headed Hereford Dwarf with Special Reference to Appendicular Skeleton	Logan M. Julian	Comp. Pathol. School of Vet. Med., Univ. of Calif.	Sept., 1956
Henry J. Adler, D.V.M. M.S.	Some Aspects of Bovine Ketosis	J. A. Dye	N.Y. State Vet. College, Cornell Univ.	June, 1957
Mohammad Zarif Durran, B.V.Sc. M.S.	Study on the Physiology of Ascaris suis and Parascaris equorum	D. W. Baker	N.Y. State Vet. College, Cornell Univ.	June, 1957
Hermann Meyer, Dr. Vet. Med.	Corticocortical Fiber Systems of the Dog's Brain	Malcolm E. Miller	N.Y. State Vet. College, Cornell Univ.	June, 1957
Lyle E. Hanson, Ph.B. D.V.M. M.S.	Pathogenesis of Virus Hepatitis of the Duck	Joseph O. Alberts	Vet. Pathol. and Hygiene, Univ. of Ill.	June, 1957
Frank Sauer, D.V.M. M.S.	Studies of the Oxygen Uptake by Liver Homogenates from Normal and Ketotic Cows	Harvey H. Hoyt	College of Vet. Med., Univ. Of Minn.	June, 1957
Nelson B. King, B.S. D.V.M. M.S.	Observations on Brucellosis	Walter G. Venake	Dept. of Vet. Med., Ohio State Univ.	March 1957
Richard Redding, D.V.M. M.Sc.	Spread of the Excitation Wave in the Ovine Left Ventricle	C. Roger Smith	Vet. Physiology & Pharmacology College of Vet. Med., Ohio State Univ.	June, 1957
Gerald D. Goetsch, D.V.M. M.S.	Studies on the Production and Treatment of Experimental Ketosis of Ruminants	W. R. Pritchard	Dept. of Vet. Sc., Purdue Univ.	June, 1957
*Charles H. Bridges, D.V.M. M.S.	Histopathological Diagnosis of Leptospirosis	H. A. Smith	Vet. Pathol. School of Vet. Med., Texas A. & M. College	Jan. 1957

\*AVMA Research Fellow

**Anaplasmosis Bibliography Available**

Through the courtesy of Dr. Melvin H. Knisely, chairman, Department of Anatomy, Medical College of South Carolina, a comprehensive bibliography on anaplasmosis is available to the veterinary profession. Prepared by a graduate student, Norman L. Garlick, B.S., D.V.M., the bibliography covers published material on the subject up to 1952. This in-

formation will be of value to research workers and students, and will be furnished free of charge to all who request it while the supply lasts. Direct requests to: Dr. Melvin H. Knisely, Chairman, Department of Anatomy, Medical College of South Carolina, 16 Lucas St., Charleston 16, S. Car.

Dr. Norman L. Garlick is now with the A.D.E. Division, U.S.D.A., ARS, as a veterinary livestock inspector in Hanford, Calif.

**Animal Care Panel Meets**

The eighth annual meeting of the Animal Care Panel will be held in San Francisco, Calif., at the Bellevue Hotel, Nov. 7-9, 1957.

The following subjects will be discussed: pathogen-free animals, pest control in animal quarters, the care and housing of laboratory animals (motion picture), a purchasing agent's view of animal colony-procurement problems, the training of animal technicians in Great Britain, studies on the development and management of inbred mouse strains.

Among other subjects on the tentative program are: practical establishment and maintenance of salmonellosis-free mouse colonies, latent infections in mice, the breeding of special strains of guinea pigs, guinea pig nutrition, care and management of opossums and their use in pediatric research, the Western burro in biological research, the production and use of Beagles in radiobiological research, and studies of normal and abnormal sexual behavior in cats and the implications in the management of a breeding colony.

Studies on stress and transportation of laboratory animals—bacteriological, pathological, and biochemical—will also be presented.

**AMONG THE STATES AND PROVINCES****California**

Dr. Willard D. Ommert (center), president-elect of the Southern California V.M.A., receives the key to the Association's new mobile unit from Dr. Fred P. Sattler (right), chairman of the Public Relations Committee. Dr. Fred Meisinger (left), a member of the PR Committee, was responsible for equipping the unit.

This completely equipped Volkswagen Kombi, which will provide better veterinary service to the public in Southern California, will be used at dog shows, rabies clinics, exhibits, and in the civil defense program.

The unit was financed by the rabies clinics in which the Association participates, and to which the members donate their time with proceeds going to the Association. The public response to this unit has been extremely favorable.

**Southern California V.M.A.**—The representative council of the Southern California V.M.A. held its monthly meeting on June 26, 1957.

Dr. Ommert discussed race track problems related to veterinary medicine, and Dr. Dean reported on the bills considered by this session of the legislature.

The question as to the degree the S.C.V.M.A. should participate with a commercial concern on future programs was considered at great length by the council. It voted that, since the council already had the authority to approve or to disapprove any association program, each future proposal would be considered separately.

The Southern California V.M.A. voted unanimously to invite the California State V.M.A. to hold its 1959 meeting at the Miramar Hotel in Santa Monica.

• • •

**Governor Signs State Rabies Law.**—Governor Knight signed into law Senate Bill No. 1231, which will require the vaccination of all dogs against rabies. Those concerned with public health attribute the successful passage of the bill to the hundreds of letters in favor of the bill sent to the governor.

• • •

**S.C.V.M.A. to Test Amount of Radiation from X-Ray Equipment.**—In an effort to determine the degree of exposure resulting from X-ray equipment used by its members, the Southern California V.M.A. has made arrangements with the California State Department of Public Health for an on-the-spot survey, beginning in September, in the small animal hospitals of its members.

Each veterinarian will be given a film badge which he will wear for two weeks as he carries on his normal practice. At the end of that period, an examination of the film badges will indicate the amount of radiation and the hazard, if any, to the veterinarian working with x-ray equipment.

Each veterinarian will receive an individual report of the findings, as well as recommendations for the proper installation and handling of his equipment.

This service was used recently by the dentists of California with much success.

#### Iowa

**Cedar Valley V.M.A.**—The meeting of the Cedar Valley V.M.A. was held June 10, 1957, in Waterloo, Iowa.

The following officers were elected for the coming year: Drs. Jack Coyne, Reinbeck, president; John Guldner, Waterloo, vice-president; and Andrew Cotten, Grundy Center, secretary-treasurer.

s/ANDREW COTTEN, Secretary.

#### Kansas

**Kansas City V.M.A.**—The July meeting of the Kansas V.M.A. convened July 16, 1957, in the Exchange Hall, Kansas City, Mo.

Dr. Earl F. Huffman, manager of professional relations at Armour Laboratories, discussed the "Use of Adrenocortical Hormones in Veterinary Practice," in the large animal portion of the program.

s/R. E. GUILFOIL, Secretary.

#### Kentucky

**South Central Kentucky V.M.A. Women's Auxiliary.**—The spring meeting of the Women's Auxiliary to the South Central Kentucky Veterinary Medical Association met at the Mammoth Cave Hotel, Mammoth Cave, on May 13, 1957, in conjunction with the South Central Kentucky V.M.A. meeting. Eighteen members participated in the business and social events.

s/MRS. G. W. FREAS, Secretary.

#### Mississippi

**State Association.**—The fifty-first annual convention of the Mississippi State V.M.A. was held at the Hotel Heidelberg in Jackson, July 14-16, 1957.

Among the speakers who addressed the Association (and their topics) were: Drs.

H. S. Bryan—Leptospirosis in Large Animals, and Bovine Mastitis; W. W. Armistead, president-elect of the AVMA.—Your Stake in the AVMA, and Practical Veterinary Plastic Surgery; R. J. Beamer—Canine Surgical Procedures, and Feline Surgery, Restraint, Medication, and Anesthesia.

Drs. Lewis J. Pate and Vernon D. Chadwick spoke on the Brucellosis Eradication Program, and Dr. John R. Dick presented illustrated talks on Problems in Swine Practice. Senator Earl Evans was the guest speaker at the Association's informal banquet, held on the roof of the hotel, July 15.

The officers for the coming year are: Drs. Jack B. Ross, Jackson, president; James W. Patterson, Columbia, president-elect; Wynon C. Stewart, Picayune, vice-president; and Harvey F. McCrory, State College, secretary-treasurer.

#### Michigan

**Officers of State Association.**—At the annual meeting of the Michigan State V.M.A., June 27, the following officers were elected: Stephen R. Kelly, Detroit, president; Charles



Dr. Stephen R. Kelly

H. Coy, Hillsdale, president-elect; William Mackie, Lapeer, first vice-president; Alvin R. Wingerter, Big Rapids, second vice-president; and Robert Jewell, Sault Ste. Marie, third vice-president.

More than 300 Michigan veterinarians and their wives attended the combined business-educational meeting.

\* \* \*

**Veterinarian of the Year.**—Dr. Edward K. Sales, director of the small animal clinic and head of the Department of Surgery and Medicine at Michigan State University, was named a



Dr. Edward K. Sales

Michigan's "Veterinarian of the Year" at the seventy-fifth annual meeting of the Michigan State Veterinary Medical Association in Detroit. Professor Sales, a member of the M.S.U. staff since 1919, was cited for his outstanding contributions to the field of veterinary medical education.

#### New Brunswick

**Joint Conference of Maritime Veterinarians.**—The eighth annual joint conference of maritime veterinary associations was held at Mount Allison University, Sackville, on June 25-27, 1957.

The program included the following speakers and subjects: Drs. Charles A. Mitchell, president, C.V.M.A., Hull, Que.—Greetings from the Canadian V.M.A.; C. K. Roe, Ontario Veterinary College, Guelph—Gastroenteric Diseases of Swine; Edith Williams, Toronto, and Jean Rumney, Hamilton—Some Aspects of Small Animal Practice; C. W. Brown, Truro, N.S.—Bovine Regional Anaesthesia; L. J. Dolan, Port Elgin, D. L. T. Smith, Guelph, and C. K. Roe, Guelph—Panel on Large Animal Diseases; H. Konst, Animal Diseases Research Institute, Hull—Considerations in the Diagnosis of Tuberculosis, Johne's Disease, and Glanders; D. L. T. Smith—Differential Diagnostic Features of Diseases in Sheep; K. F. Wells, veterinary director general, Health of Animals Division, Ottawa—The Role of the Practitioner in Federal Control Policies; and Douglas Mitchell, Animal Diseases Research Institute, Hull—Some Aspects of Bovine Infertility.

Mr. John Fisher, executive director, Canadian Tourist Association, spoke at the banquet, followed by an evening of informal entertainment under the direction of Drs. George Fisher and

Carl Jarvis. The ladies had a separate formal program.

s/J. S. FRANK, Resident Secretary.

#### North Carolina

**Women's Auxiliary.**—The annual meeting of the Women's Auxiliary was held in conjunction with the North Carolina V.M.A. at the Grove Park Inn, Asheville, June 25-27. The guest of honor was Mrs. A. E. Coombs, of Skowhegan, Maine, president of the Women's Auxiliary to the AVMA.

Among the charter members present were: Mrs. M. M. Leonard, Asheville; Mrs. N. B. Tyler, Raleigh; and Mrs. J. I. Neal of Southern Pines.

It was voted to contribute \$25 to the AVMA Research Fund; \$25 to the Library Fund, School of Veterinary Medicine, University of Georgia; \$25 to the Library Fund, School of Veterinary Medicine, Oklahoma A. & M. College; \$10 to the General Loan Fund, and \$5 to the *Auxiliary News*. Mrs. J. W. Parcher of Hendersonville was appointed delegate and Mrs. J. I. Neal, alternate, to the AVMA Auxiliary meeting at the AVMA convention in Cleveland.

Officers for the coming year are: Mrs. C. E. Young, Mocksville, president; Mrs. W. O. Slappy, Fayetteville, vice president; and Mrs. J. I. Cornwell, Asheville, secretary-treasurer.

s/Mrs. J. I. CORNWELL, Secretary.

#### South Carolina

**State Association.**—The forty-sixth annual meeting of the South Carolina Association of Veterinarians was held at the Fort Sumter Hotel, Charleston, on June 20-22, 1957.

Speakers on the program included Drs. W. W. Armistead, dean, College of Veterinary Medicine, Texas A. & M. College, and president-elect of the AVMA; George Armstrong, Charlotte, N. Car.; David Bartlett, American Breeders Association, Chicago; T. E. Brown, president, South Carolina Association, Spartanburg; R. W. Carter, state-federal director, Disease Eradication Program, Columbia; John Dick, Fort Dodge Laboratories, Iowa; Frank G. Fielder, veterinary director, Shering Corp., New York City; H. L. Frieze, Gaffney; Glen Lawhon, Jr., Hartsville; W. K. Magill, Chester; George Moore, Walterboro; Joseph Skelley, University of Pennsylvania, Philadelphia; Otto Strock, Charleston; Mayor Wm. McG. Morrison, Charleston; Wm. M. McCord and Robert Walton, Medical College of South Carolina, Charleston.

The following officers were elected for the ensuing term: Drs. T. E. Brown, Spartanburg, president; George Smith, president-elect; T. M. Rhodes, Charleston Heights, vice-president; and Worth Lanier, York, secretary-treasurer.

The next meeting, June, 1958, will be held in conjunction with the Georgia Association at the Bon Air Hotel in Augusta.

## Utah

**Dr. A. K. Kuttler Assumes Post of State Veterinarian.**—Dr. A. K. Kuttler, an authority on animal disease eradication, particularly in work on brucellosis, retired from the U.S.D.A. on June 30, 1957. He was director of state-federal programs in Utah.

Immediately after retiring from the U.S.D.A., he assumed duties as state veterinarian of Utah.

During the time that he was in charge of the national brucellosis program, the entire states of Maine and New Hampshire were declared modified certified brucellosis-free, and work leading to certification was in progress in most of the states. In the reorganization of the department in 1954, he was made chief of the brucellosis section of the Animal Disease Eradication Branch. Dr. Kuttler also took an active part in the establishment of the National Brucellosis Committee.

## Wyoming

**State Association.**—The summer meeting of the Wyoming V.M.A. was held at Lander, June 15-17, 1957.

Dr. Alan Tench, with the ARS at Lander, compared the ethics of the British and American veterinary professions. Dr. R. C. Knowles, also with the ARS at Cheyenne, described the effects of ionizing radiation on animals. Dr. G. H. Good, state veterinarian, covered the current scabies situation in sheep. Mr. Carl Gilbert, Department of Research Chemistry, University of Wyoming, described lupine poisoning in sheep. Mr. C. E. Bell, a Lander banker, spoke on the importance of the veterinarian to the banker, once he becomes aware of the type of assistance the veterinarian can offer.



Dr. G. H. Good (left), Wyoming State Veterinarian; Dr. E. S. Norton, president, Wyoming V.M.A.; Dr. Wayne O. Kester, president, AVMA; and Dr. David O. Manley, chief, Wyoming ARS.

Other speakers and their subjects were: Dr. Wayne O. Kester, president of the AVMA—AVMA Items of Interest and Approach to Equine Practice; Dr. F. K. Bracken, Washington State College—Common Poisoning of

Farm Animals and a New Look at Some Old Liver Lesions; Dr. E. E. Saulmon, district supervisor of the Animal Disease Eradication Branch, ARS—Activities of the A.D.E. program; and Dr. C. J. York, Pitman-Moore Co., Indianapolis—New Developments in Canine Distemper and Rhinotracheitis.

The new officers elected for the coming year are: Drs. J. E. Ketcham, Cheyenne, president; John A. Wilson, Sheridan, vice-president; and J. F. Ryff, Laramie, secretary-treasurer.

## Texas

**Baylor's New Laboratory of Physiology Attracts Veterinarians.**—Four members of Veterinary college faculties attended a summer workshop at Baylor University College of Medicine



The veterinarians who attended the summer workshop at Baylor University are (left to right)—Dr. Verner L. Johnson, College of Veterinary Medicine, State College of Washington, Pullman; Dr. Loyal C. Payne, College of Agriculture, University of Nebraska, Lincoln; Dr. L. L. Nangeroni, New York State Veterinary College, Cornell University, Ithaca; and Dr. Donald H. Will, Colorado A. & M. College, Fort Collins.

in Houston on "Classical Physiology with Modern Instrumentation." The course has attracted men and women from Canada, Brazil, Colombia, and the United States—veterinarians, doctors of medicine, engineers, lecturers, and teachers.

The photograph was taken with the physiograph, a new instrument which allows a new approach to physiological study.

## DEATHS

Star indicates member of AVMA

★**Chester F. Clark** (MSU'29), 57, dean of the College of Veterinary Medicine, Michigan State University, died on July 28, 1957, after an illness of several months due to generalized melanomatosis, and only a short time before he was to be retired, at his request, and become dean emeritus.

Born Oct. 31, 1899, at Fitchburg, Mass., Dr. Clark received his preveterinary education at Fitchburg High School and at Massachusetts

State Agricultural College which he attended from 1919 to 1921. He then enrolled for a year at the former U. S. College of Veterinary Surgeons in Washington before entering Michigan State where he received his D.V.M. degree in 1929.

Following graduation, he became a member of the animal pathology staff at the college and had been a full-time faculty member ever since except for a three-year period, 1946-1949, when he served as state veterinarian of Michigan.



Dr. Chester F. Clark

Dr. Clark was made head of the Department of Surgery and Medicine in 1949 and was named dean following the death of Dr. Claude S. Bryan in 1951. He directed the planning and reorganization in connection with the veterinary college's move into its new and greatly enlarged plant, then under construction.

Before his appointment to the deanship, Dr. Clark had spent more than 20 years working with Michigan veterinarians, both as a member of the M.S.U. staff and as state veterinarian. His research activities centered in the field of dairy cattle diseases, especially brucellosis and reproductive disorders. He was the author of a number of articles on these conditions.

Dr. Clark joined the AVMA in 1936 and had served on a number of its standing and special committees including the Committee on Legislation of which he had been chairman for the past two years. In 1949, he was general secretary of the local arrangements committee for the Detroit AVMA Convention. He was also a frequent contributor to its meeting programs and to those of other veterinary and scientific associations.

He was also a member of the U. S. Livestock Sanitary Association, the Conference of Official

Workers in Animal Disease Research, Association of Chief Livestock Sanitary Officials, Phi Zeta, Alpha Psi, and Sigma Pi.

Dr. Clark is survived by his widow, Mrs. Muriel Hoover Clark whom he married in 1930, and three children, a son, Dr. John H. Clark, a practicing veterinarian in the Detroit area; a daughter, Marjorie and a son Andrew.

[Announcement of the appointment of Dr. W. W. Armistead as Dean Clark's successor appeared in the August 15 issue.—Ed.]

★**George F. Barton** (ONT '32), 50, Chilliwack, B.C., Canada, died July 4, 1957, after a boating accident at Kamloops, B.C. Dr. Barton is survived by his widow, Bernice.

**Leo D. Bashore** (CIN '15), 68, Paulding, Ohio, died June 5, 1957. He had been seriously ill for four months. Dr. Bashore, a World War I veteran, had been a veterinarian for over 40 years.

He is survived by his widow, a daughter, and two sons.

**S. J. Collins** (ONT '91), 87, Reedsburg, Wis., died March 15, 1957. Dr. Collins is survived by his widow, one daughter, and one son.

**Wynter C. Gruber** (OSU '09), 74, Nashville, Tenn., died June 22, 1957. Dr. Gruber suffered a heart attack while performing his veterinary duties as a poultry inspector for the Agricultural Marketing Service, U.S.D.A.

★**Ralph L. Gangarosa** (Ganis) (COR '40), 38, Gordon, Neb., died July 3, 1957, in a head-on automobile collision near Sydney, Neb.

Dr. Gangarosa was the last surviving member of his immediate family. His wife and four children perished in a fire in their home in June, 1954.

He was a member of Phi Zeta.

**Eugene F. Pile** (KSC '16), 65, Lexington, Ky., died following a heart attack. He was a native of Kansas but had lived in Kentucky for the past 18 years.

**Gilbert S. Kirby** (STJ '19), 64, Warsaw, Mo., died May 16, 1957, following several months illness. Dr. Kirby, a World War I veteran, taught at three rural schools prior to entering the profession.

He is survived by his widow, Kathryn, and two daughters.

★**Harvey H. Seely** (CVC '18), 74, Jerseyville, Ill., died June 15, 1957. Dr. Seely was affiliated with the Twelfth International Veterinary Congress, Jerseyville Lodge, A.F. & A.M., the Loyal Order of Moose, Scottish Rites Mississippi Consistory, and the Ainaad Temple, East St. Louis.

He is survived by his widow, Tula, a brother, and a sister.

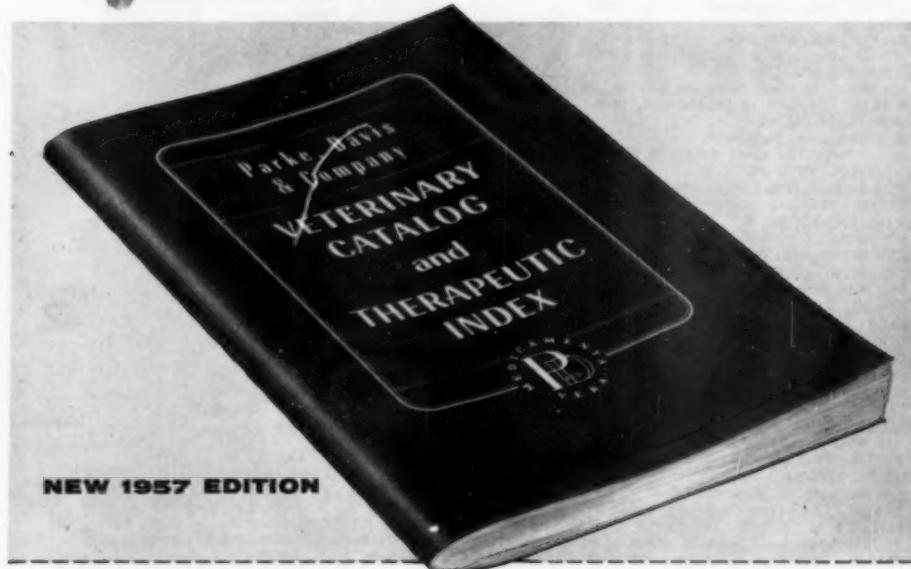
**Robert S. Tillie** (ISC '16), Muscatine, Iowa, died April 26, 1957. He conducted his practice in Muscatine since the time of his graduation in 1916.



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## ORGANIZATION SECTION

### APPLICATIONS

#### Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

- ABBEY, HAROLD K.  
P.O. Box 92, Lancaster, Ont.  
D.V.M., Ontario Veterinary College, 1947.  
ABY, E. S.  
Box 397, Glendive, Mont.  
D.V.M., Colorado State University, 1950.  
COLANDO, ANDREW C.  
160 Manor Dr., Red Bank, N. J.  
V.M.D., University of Pennsylvania, 1948.  
DOWNIE, NIALL F.  
4409 Kingsway, Burnaby I.  
M.R.C.V.S., The Royal Veterinary College, 1952.  
FERGUSON, FREDERICK G.  
1702 S. Dort Hwy., Flint, Mich.  
D.V.M., Michigan State University, 1943.  
JULIA, THEODORE R.  
5 Vose St., Waterville, Maine.  
D.V.M., Ontario Veterinary College, 1955.  
KOESTNER, ADALBERT  
986 Hilo Lane, Columbus, Ohio.  
D.V.M., University of Munich, 1951.  
ORLOFF, PAUL J.  
6 Pleasant Court, Waterville, Maine.  
D.V.M., Michigan State University, 1956.  
PEARSON, KERMIT C.  
3745 Harrison, Butte, Mont.  
D.V.M., Washington State College, 1949.  
PHILLIPS, HOWARD C.  
935 West Broadway, Gardner, Mass.  
D.V.M., Michigan State University, 1946.  
PHIPPS, EDWARD C.  
740 Sandusky St., Fostoria, Ohio.  
D.V.M., Ohio State University, 1934.  
RYDER, J. G.  
52 Townsend Ave., Norwalk, Ohio.  
D.V.M., Ohio State University, 1952.  
SAKALAUSKAS, JULIUS  
17270 60th Ave., Surrey Centre, B. C.  
D.V.M., Veterinary Academy of Kansas, 1942.

#### Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (\*) after the name of a school indicates that all of this year's graduates have made application for membership.

#### First Listing

#### University of California

- BOMAN, ALF E., D.V.M.  
1950 Oleander Ave., Merced, Calif.  
Vouchers: M. J. Silver and A. J. Eisenhower.

#### University of Georgia

- ATWELL, JOHN K., D.V.M.  
Rt. 1, China Grove, N. Car.  
Vouchers: S. S. Kreus and A. M. Mills.  
DODDS, A. J., D.V.M.  
1609 Duncan Ave., Chattanooga, Tenn.  
Vouchers: G. P. Hatchett, Sr., and G. P. Hatchett, Jr.  
KAMENETZ, LEONARD, D.V.M.  
414 North East Ave., Waukesha, Wis.  
Vouchers: P. L. Piercy and A. M. Mills.  
MAXWELL, ELLEN J., D.V.M.  
Sarahlyn Farms, Watkinsville, Ga.  
Vouchers: P. L. Piercy and F. A. Hayes.  
STONE, HENRY D., D.V.M.  
Rt. 1, Union Bridge, Md.  
Vouchers: S. S. Kreus and A. M. Mills.  
SWAHN, FREDERICK H., D.V.M.  
R.D. 1, Whiteford, Md.  
Vouchers: M. Rohleder and P. L. Piercy.  
WEISS, JAMES D., D.V.M.  
196 Grayrock Pl., Stamford, Conn.  
Vouchers: A. M. Mills and E. W. Causey.  
WILKINSON, JAMES C., D.V.M.  
R.F.D. 2, Chilhowie, Va.  
Vouchers: J. R. Fenik and C. W. Henry.

#### University of Illinois

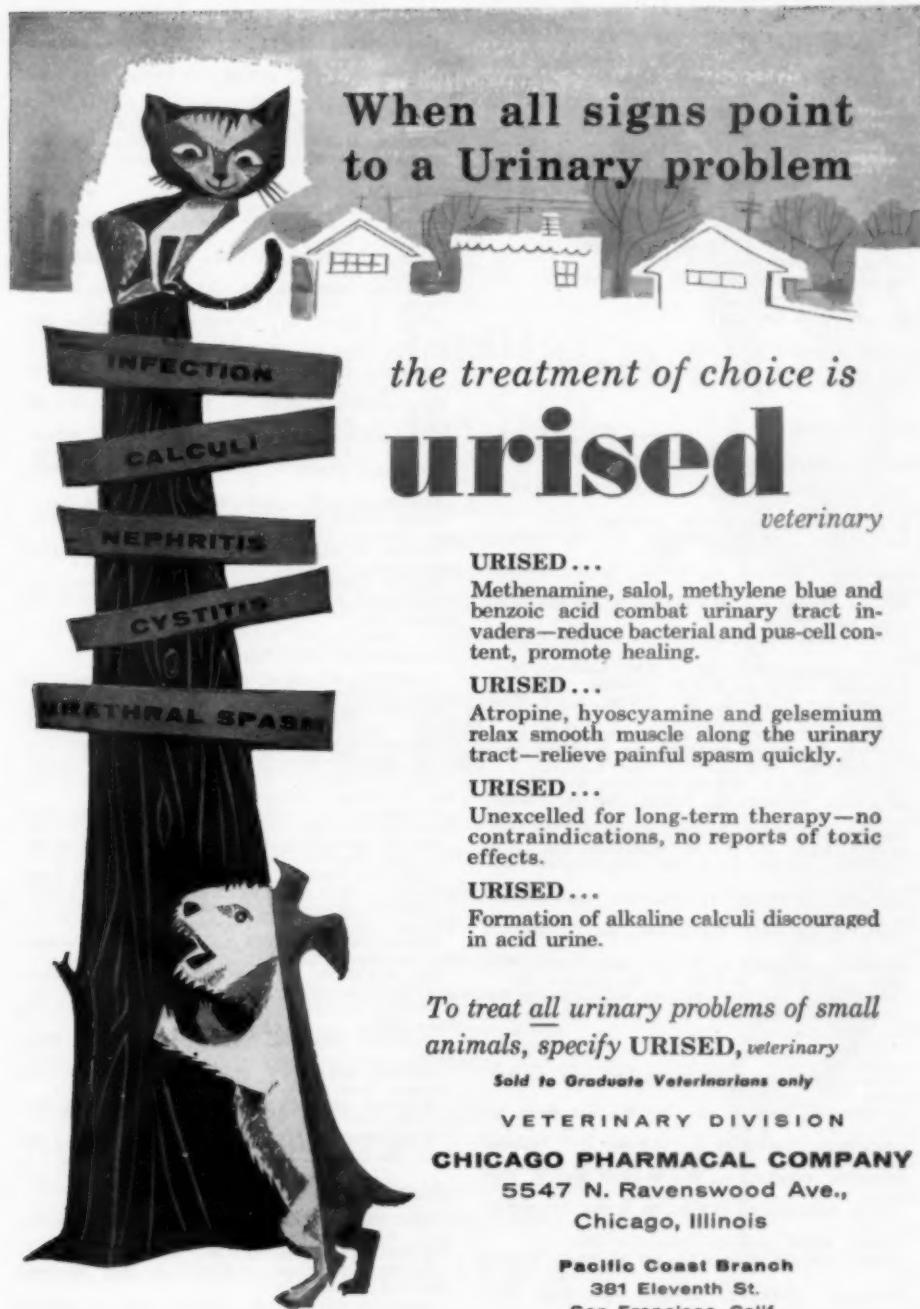
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- New Mexico Veterinary Medical Association.** Annual meeting. Albuquerque, Sept. 9-10, 1957. William E. Kraus, 3018 Rio Grande Blvd., N.W., Albuquerque, president.
- Washington State Veterinary Medical Association.** Annual meeting. Monticello Hotel, Longview, Sept. 9-10, 1957. William F. Harris, 1102 E. Main St., Puyallup, Wash., secretary.
- New York State Veterinary Medical Society.** Annual meeting. Hotel Statler, Buffalo, Sept. 11-13, 1957. Joan S. Halat, 803 Varick St., Utica, secretary.
- Northern Illinois Veterinary Medical Association.** Fall meeting. Rockford, Sept. 18, 1957. J. G. Hardenbergh, 121 Bridge Street, Rockton, Ill., secretary-treasurer.
- South Dakota Veterinary Medical Association.** Annual convention. Hotel Cataract, Sioux Falls, Sept. 19-20, 1957. J. L. Noorday, Marion, S. Dak., secretary.
- District of Columbia Veterinary Medical Association.** Third annual all-day meeting. Sternberg Auditorium, Walter Reed Army Medical Center, Oct. 1, 1957. Wm. I. Gay, 5200 Chandler St., Bethesda 14, Md., secretary.
- New England Veterinary Medical Association.** Annual meeting. Equinox House, Manchester, Vt., Oct. 6-9, 1957. C. Lawrence Blakely, 180 Longwood Ave., Boston, Mass., secretary.
- Purdue University.** Annual short course for veterinarians. Purdue University, West Lafayette, Ind., Oct. 9-11, 1957. L. M. Hutchings, secretary.
- Florida State Veterinary Medical Association.** Annual meeting. Fort Harrison Hotel, Clearwater, Oct. 13-15, 1957. Robert P. Knowles, 2101 N.W. 25th Ave., Miami 42, Fla., secretary.
- University of Missouri.** Annual short course for graduate veterinarians, Oct. 14-15, 1957, School of Veterinary Medicine, University of Missouri, Columbia. Cecil Elder, chairman.
- Pennsylvania State Veterinary Medical Association.** Annual meeting. Hotel Brunswick, Lancaster, Oct. 16-18, 1957. Raymond C. Snyder, N. W. Corner Walnut St. and Copley Rd., Upper Darby, secretary.
- Texas Veterinary Medical Association.** Annual meeting. Baker Hotel, Dallas, Oct. 16-18, 1957. Paul B. Blunt, 712 Maverick Bldg., San Antonio, Texas, secretary.
- Eastern Iowa Veterinary Association.** Annual meeting. Hotel Sheraton-Montrose, Cedar Rapids, Oct. 17-18, 1957. F. E. Brutman, Traer, Iowa, secretary.
- Illinois, University of.** Annual veterinary conference and short course. School of Veterinary Medicine, University of Illinois, Urbana, Oct. 17-18, 1957. L. E. Boley, chairman.
- Southern Veterinary Medical Association.** Annual meeting. Hotel Roanoke, Roanoke, Va., Oct. 27-30, 1957. A. A. Husman, P. O. Box 91, Raleigh, N.C., secretary.
- Cornell University.** Nutrition conference. Cornell University, Ithaca, N.Y., Oct. 31-Nov. 1, 1957. J. K. Loosli, Stocking Hall, Cornell University, Ithaca, N.Y., chairman.
- Animal Care Panel.** Annual meeting. Bellevue Hotel, San Francisco, Calif., Nov. 7-9, 1957. R. J. Flynn, Box 299, Lemont, Ill.
- Pennsylvania, University of.** Annual conference for veterinarians. School of Veterinary Medicine, Philadelphia, Jan. 7-8, 1958. M. W. Allam, dean.
- Cornell University.** Annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 8-10, 1958. W. A. Hagan, dean.
- Kansas Veterinary Medical Association.** Annual conven-
- tion. Hotel Broadview, Wichita, Jan. 12-14, 1958. K. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.
- Intermountain Veterinary Medical Association.** Annual meeting. Hotel Utah, Salt Lake City, Jan. 16-18, 1958. R. A. Bagley, 4600 Creek View Dr., Murray, Utah, secretary.
- Minnesota Veterinary Medical Association.** Annual meeting, St. Paul, Jan. 20-22, 1958. B. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn.
- North Carolina State College.** Conference for veterinarians. North Carolina State College, Raleigh, Jan. 28-31, 1958. C. D. Grinnells, chairman.
- Oregon Veterinary Medical Association.** Winter meeting. Portland, Jan. 31-Feb. 1, 1958. Edward L. Holden, P. O. Box 445, Oswego, secretary.
- Regularly Scheduled Meetings**
- ALABAMA—Central Alabama Veterinary Association,** the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.
- Jefferson County Veterinary Medical Association,** the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.
- Mobile-Baldwin Veterinary Medical Association,** the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.
- ARIZONA—Central Arizona Veterinary Medical Association,** the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.
- Southern Arizona Veterinary Medical Association,** the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.
- CALIFORNIA—Alameda Contra Costa Veterinary Medical Association,** last Wednesday of each month. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.
- Bay Counties Veterinary Medical Association,** the second Tuesday of each month. Maurice L. Boevers, 3394 Mt. Diablo Blvd., Lafayette, Calif., secretary.
- Central California Veterinary Medical Association,** the fourth Tuesday of each month. R. B. Barssaleau, 2333 E. Mineral King, Visalia, Calif., secretary.
- Kern County Veterinary Medical Association,** the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.
- Mid-Coast Veterinary Medical Association,** the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.
- Monterey Bay Area Veterinary Medical Association,** the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.
- North San Joaquin Valley Veterinary Medical Association,** the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.
- Orange Belt Veterinary Medical Association,** the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.
- Orange County Veterinary Medical Association,** the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.
- Peninsula Veterinary Medical Association,** the third Monday of each month. R. C. Lawson, 4040 El Camino, Palo Alto, Calif., secretary.
- Redwood Empire Veterinary Medical Association,** the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.
- Sacramento Valley Veterinary Medical Association,** the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.
- San Diego County Veterinary Medical Association,** the

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# TRILAFON Dosage Guide

Animal	Oral	Intravenous	Intramuscular
DOG	4 mg./10 lb. b.i.d.	5 mg./20 lb.	5 mg./20 lb.
SWINE			10 mg./100 lb.
ADULT BOVINE		75-125 mg.	100-150 mg.
CALVES & HEIFERS		Adjust adult dosage according to body weight and degree of tranquilization required.	

Dosage should be adjusted to the size of the animal and degree of tranquilization required.

#### onset of effect

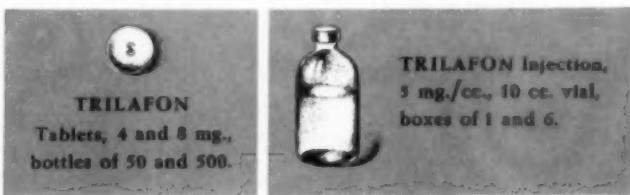
In dogs, when administered as recommended, TRILAFON Tablets are effective in 45 minutes to one hour. TRILAFON Injection, intramuscularly, is effective in 20 to 30 minutes and intravenously in 10 to 20 minutes.

In swine and the bovine TRILAFON Injection is effective intramuscularly in 30 to 45 minutes, and intravenously in 10 to 15 minutes.

Duration of action depends upon the species, size, temperament and dose administered. Effects may last 6 hours or longer.

#### Packaging

##### SMALL ANIMALS



##### LARGE ANIMALS



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fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

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Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 3060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarskjold, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington. Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodson's Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified

monthly. T. R. Geci, 109B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 300-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 May St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at

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the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

East Central Iowa Veterinary Medical Society, the second Tuesday of every month. Dr. W. T. Rugger, Oxford, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KANSAS—Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Associa-

tion, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison Sts., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Piegge, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hight Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mansa in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

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McEachran, D.: Am. Vet. Rev. 3:108 (Jan.) 1879.

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"Some cases [of anthrax] failed to respond to penicillin therapy. Terramycin was then used concurrently with penicillin and the response was prompt and effective."

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"To date, 200 cases have been treated with Terramycin alone or concurrently. Terramycin intramuscularly plus penicillin, streptomycin and penicillin alone, in the majority of cases Terramycin alone yielded the most favorable results." Miller, V. W., et al. J. Am. Vet. Med. Assoc. 149:104 (April) 1966.

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Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College. Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twain Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association.

the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

Stark County Veterinary Association, the second Monday of each month. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forrest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

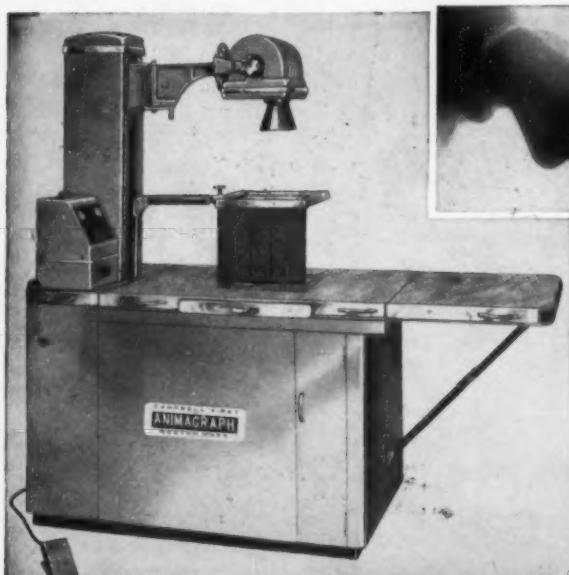
PENNSYLVANIA—Del-High Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary. Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

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Her story began on a night very long ago when, as a Quaker girl in Nantucket, Maria Mitchell discovered a comet—and got a gold medal worth 20 ducats from the Danish King.

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That was because they didn't know Maria Mitchell. At 12 she could regulate a ship's chronometer; at 17 she understood Bowditch's "Practical Navigator" and was studying science in self-taught French, German and Latin. In time she would become the first woman member of the American Academy of Arts and

Sciences, the first woman astronomy professor—in Matthew Vassar's Female College—and a member forever of New York University's Hall of Fame.

Moreover, all her adult life she was to work with growing success in the crusade to make American women free.

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REFERENCES: 1. Mosier, J. E.: Vet. M. 50:605 (Nov.) 1955.  
2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956.  
3. Pollock, S.: J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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Because of the interest in veterinary radiology, a case history and radiographs depicting a diagnostic problem are usually published in each issue.

***Make your diagnosis from the picture below—then turn the page.***

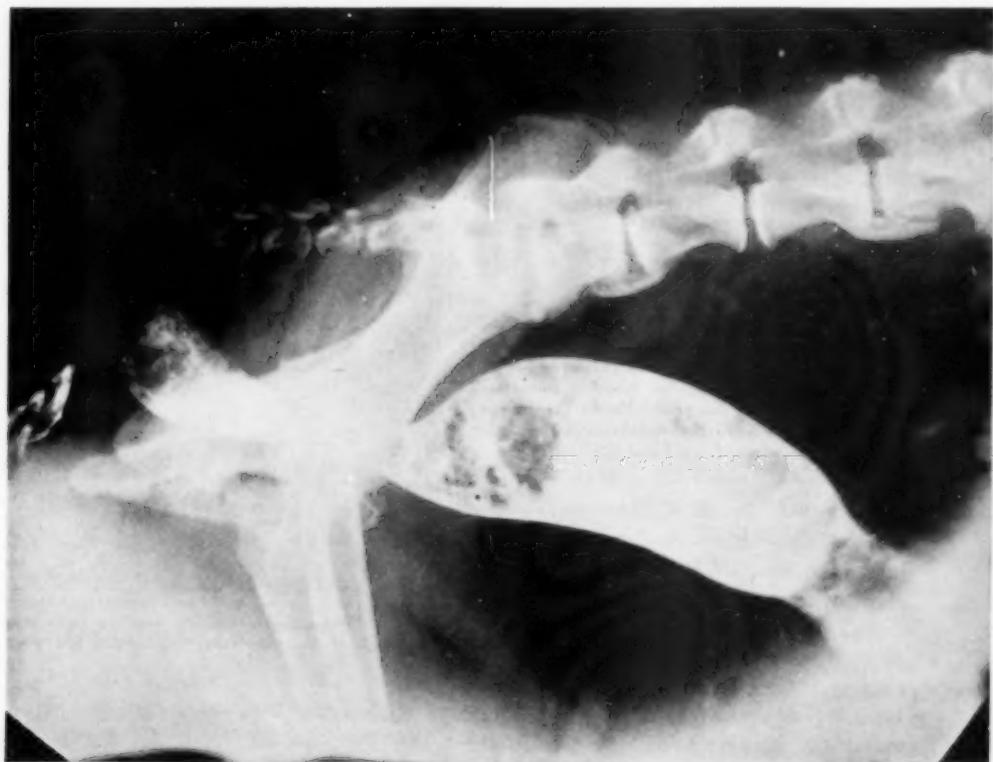


Figure 1

***History.***—A spayed Irish Setter, 10 years old, developed chronic constipation. During a period of several weeks, straining at defecation increased. On digital examination a firm, fixed mass could be reached, dorsal to the rectum. Barium solution was injected per rectum into the large bowel and a radiograph, lateral view, was taken.

***(Diagnosis and findings are reported on next page)***

## Here Is the Diagnosis

(Continued from preceding page)

**Diagnosis.**—An egg-shaped mass in the pelvis which compresses the rectum dorsally, also an ossifying spondylitis of at least three vertebrae (fig. 2). Histologically, this proved to be a sarcoma of undetermined type.

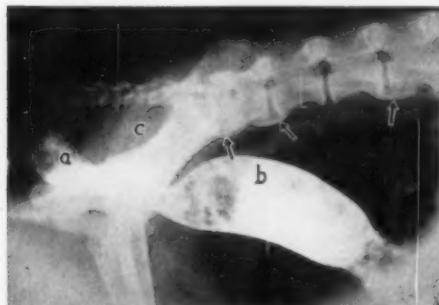


Fig. 2—Radiograph, lateral view, pelvic and lumbar regions. The rectum (a), and colon (b), has been outlined with barium solution. The bowel is compressed by the tumor (c) in the pelvis. Notice the ossifying spondylitis (arrows) on the vertebrae (L-7 and S-1, L-6 and 7, L-4 and 5).

**Comments (Dr. E. E. Ruebush).**—One month following surgical removal of the mass, the animal seemed healthy and all signs of constipation had disappeared.

**Discussion of the Histopathology (Dr. T. C. Jones).**—The tumor is composed of cells that form cords which tend to interlace. When several cords converge, they produce a whorled effect (fig. 3). In the more vascular areas, the cells are arranged in a concentrically laminated pattern around capillaries.

The tumor is composed of spindle-shaped cells having nuclei of various shapes. Some nuclei are knobby, potato-shaped, and

vesicular. Others are fusiform, ovoid, or almost spherical. Most are vesicular and have one nucleolus. The cytoplasm is eosinophilic, fibrillar, and streams out from each pole of the nucleus giving the cell its spindle shape.

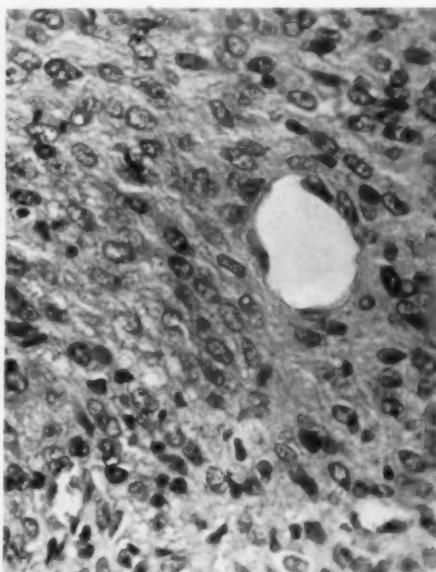


Fig. 3—Histological section showing the pattern of the tissue of the neoplasm. Hematoxylin and eosin. x 375.

**Histological Diagnosis.**—Sarcoma of undetermined type from the pelvic cavity of a dog. (In view of the difficulty in removal and the microscopic analysis, recurrence may be expected.)

This case was submitted by Dr. E. E. Ruebush, Silver Springs, Md., and reviewed by a committee from the American Veterinary Radiological Society.

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**WASHINGTON**—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

**South Puget Sound Veterinary Association**, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

**WEST VIRGINIA**—Kyowa (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

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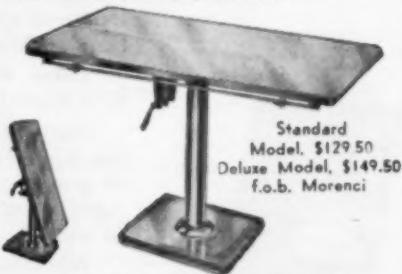
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## Radio and Television Programs at Cleveland

(Continued from adv. page 6)

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from 6:15 to 6:30 p.m.

Subject: Veterinary Medicine.  
Speaker: AVMA President, Brig. Gen. W. O. Kester.

15-minute interview with "Captain Penny" at noon, August 19.  
Subject: Women Veterinarians.  
Speaker: Dr. Jean Holzworth.

15-minute interview with "Captain Penny" at noon, August 20.  
Subject: Care and Handling of Pet Birds.  
Speakers: Dr. I. E. Altman.

WJW—15-minute interview, August 19, 11:00 to 11:15 a.m.  
Subject: Women Veterinarians and Zoo Practice.  
Speaker: Dr. Patricia O'Connor.

15-minute interview, August 20, 11:00 to 11:15 a.m.  
Subject: Radiation and Meat.  
Speaker: Colonel B. F. Trum.

15-minute interview, August 21, 11:00 to 11:15 a.m.  
Subject: Pet Maternity and Puppy Care.  
Speaker: Dr. Frank Booth.

WJW-TV (Cleveland)—15-minute interview, August 20, 9:30 - 9:45 a.m.  
Subject: Veterinary Medicine.  
Speaker: Dr. W. W. Armistead.

15-minute interview, August 21, 9:00 - 9:15 a.m.  
Subject: Veterinary Corps.  
Speaker: Brig. Gen. Wayne O. Kester.

15-minute interview, August 22, 9:30 - 9:45 a.m.  
Subject: What the Veterinarian Does for Farmers.  
Speaker: Dr. R. Anderson.

WGAR (Cleveland)—15-minute interview, August 19, 1957, 9:30 a.m.  
Subject: Veterinary Public Health.  
Speaker: Dr. C. H. Pels.

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